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ADDRESS OF THE PRESIDENT THE RELATION OF SURGEON AND HOSPITAL

ARTHUR W. ELTING, M.D.

ALBANY, N. Y.

FOR THE great honor and distinction which you conferred upon me last June in selecting me to be your presiding officer upon this occasion, may I again express my sincere gratitude and appreciation?

The preparation of the address which your president is expected to deliver has been my chief anxiety and concern during the past year. In the hope of finding some inspiration from the thoughts of my 55 illustrious predecessors, I have carefully perused their addresses.

I find that in the main they may be divided into two groups: Those in which purely surgical topics have been discussed, and those in which some of the more general problems confronting the surgical profession have been considered. It seemed to me that at the present time it might be more profitable to discuss some of the problems that belong in the latter group.

Much attention has from time to time been given to *what* should be the education and training of a surgeon, notably in the addresses of some of our more recent presidents. To this at present little, if anything, can be added. In all this thought and discussion as to *what* should be the education and training of a surgeon, very little, if any, attention has been given to *where* this can or should be accomplished. Perhaps it has been assumed that we already possess enough hospitals of high standards in which these young men could be trained, or if not that they would soon come into existence. A care-

ful survey of the hospital facilities of this country at this time demonstrates conclusively that both these assumptions are fallacious.

There are at the present time in this country comparatively few hospitals where adequate training can be had and it will require much time, thought, organization and hard work to bring the standards and administration of a sufficient number of hospitals to the high level required.

There would appear in general to be substantial agreement upon the broad principles which should govern the basic training of the surgeon as well as upon the more technical phases of the postgraduate years. These ideas, principles and discussions may be said to have been largely responsible for the initial action taken by this association three years ago which led to the formation of the American Board of Surgery. If, however, the efforts of that Board and the splendid support which it has received from the surgical profession are to achieve the desired results, we surgeons must all of us give serious thought and untiring effort to the elevation of hospital standards to a level adequate for such training. This is specifically the obligation of the leaders of American surgery, and if they do not do it, it will not be done or at best will be poorly done. They must assume the responsibility for the elevation and maintenance of the standards of hospitals so far as surgery is concerned and must not allow it to be determined by our legislatures, state or national.

It is, therefore, very important that we immediately give serious attention to the problem of our hospitals, which, in the last analysis, with their wards, operating rooms, outpatient departments and laboratories, are where the young medical man of today must be trained to become the surgeon of tomorrow.

Is it not proper and fitting that surgeons as a group should be the most interested and responsible members of the hospital staff?

Practically speaking, the essential original incentive for the development of hospitals came from the need of a place properly organized and equipped for surgery. In the early days most of a hospital's activity was connected with surgery, at a time when the other branches of the medical art were largely practiced in the home.

The demands of surgery and the financial returns to the hospital from surgical patients are largely responsible for the remarkable development of hospitals which has taken place in this country since the birth of this association 59 years ago.

Since, therefore, we of the surgical profession have been so largely responsible for the development of hospitals and since we and our patients provide such a large part of the financial income of these institutions, should it not be our privilege, as well as our duty, to take an active part in the management and direction of the professional activities of our respective hospitals?

We know in general what the training of a surgeon should be and we are the only really competent persons to direct that training and to determine to a large extent what should be the hospital's attitude toward that training.

Unless we recognize and adequately meet our responsibility, the training will be ineffective and we shall be derelict in our duty.

It is, I think, obvious that today we are living in a bureaucratic era, and it is my firm belief that the same bureaucracy, which most of us deplore, is gradually taking control of our hospitals, for even medical men are not altogether immune from this infection.

We medical men are not, by and large, good business men. As the financial structure of our hospitals has become larger and more complicated, the business man has played an increasingly more important rôle. As long as he has devoted his attention to the financial and business management, he has, on the whole, been of great assistance.

There is no occasion for any conflict between the lay group and the professional group if each will recognize and respect the knowledge and experience of the other. If, however, medical men in general, and surgeons in particular, are not on their guard they will soon cease to play the important part they should in hospital management.

Sooner than we may wish government, local, state or federal, will be called upon to greatly increase their contributions to the support of hospitals and with this will come an increasing participation of government officials in hospital administration and, if we are not prepared to guide and direct them, an increasing control of professional matters.

The most effective way to avoid this undesirable result is for us to take an ever more active interest in determining the policies and directing the professional affairs of our hospitals. To appreciate the dangers ahead of us is to be prepared to successfully meet them.

The layman has his sphere in hospital management and the professional man has his. The "no man's land" between these spheres is where both groups should seek a harmonious solution of their mutual problems.

Another of the most important hospital problems of the moment is the development of a true spirit of efficiency and economy. While a surgical department is, as a rule, a hospital's most important source of operating income, it is also a hospital's most costly activity. Hospitals in general, and surgical departments in particular, are both extravagant and wasteful, not from intent but because in our hospital relationships from third or fourth year medical student to chief of the Surgical Service we have never been taught to be or compelled to be economical. The practice of economy is never of sudden or spontaneous development but is rather the developed habit of a lifetime, a habit possessed to a remarkable degree by our forefathers but conspicuous by its absence in the American life of today.

As elder surgeons, it is our duty to see that economical principles and practices are instilled into the minds of the young from the early days in the medical school so that later on they will become the habit of their professional lives.

There is not a surgical department in any hospital in this country today in which there is not a large amount of waste of time, materials and the many

other items which make up the cost of maintenance of the department. The surgeon and the nurse are the outstanding ones who can effectively control this wasteful tendency and this can be accomplished by the development of an economic conscience in everyone who participates in the operation of the surgical department. How many of the costly whims and gadgets with which surgical departments are burdened are of any practical value? Simplification rather than complication of surgical procedure should always be our aim. Efficiency goes hand in hand with economy and true efficiency always means economy. It has long been my thought that the watchwords of a hospital should be efficiency, economy and most important of all—humanity; and who better than the surgeon can teach and practice this triad of virtues?

Another most important phase of hospital activity should be the development of a teaching atmosphere. This, of course, does already exist in a relatively small number of hospitals, especially those intimately connected with medical schools, but it does not exist in the great majority of hospitals throughout the land. Unless this teaching responsibility is recognized and the opportunities provided, how can adequate training for the surgeon of the future be had and where can young men secure the training and experience to render them eligible for the hall-mark of surgery which the American Board of Surgery proposes to place upon them?

In addition to those hospitals which already afford adequate facilities for such training, there should be many more such institutions in the near future scattered over our country. What a boon this would be to both the public and the profession and what a stimulus to the thousands of other hospitals to advance their standards. This, it seems to me, is largely the duty of American surgeons and especially of the members of this Association who in their hospital relationships represent many more than the number of adequately equipped hospitals needed today to train the surgeon of tomorrow.

From the latest report of the Council on Medical Education and Hospitals of the American Medical Association it would appear that there are only 44 hospitals in the United States which offer surgical services of three years or more. From this report it is impossible to determine in how many of these 44 hospitals the so-called intern year is included in the three years or more, but it is probable that it is included in a considerable number. This indicates how inadequate at present the surgical service in all but a comparatively few hospitals is, to give the training now required by the American Board of Surgery.

Concerned with our immediate problems of the moment, are we, as the leaders of American surgery, sufficiently aware of our duties and responsibilities for the future, so that when the torch passes from our hands it will be grasped by men better prepared than we to carry it on to their successors? Are we not devoting too much of our thought and energy to our specific personal interests and too little to the institutions we serve?

Closely associated with the foregoing, as one of the most important hospital problems, is the education and training of the nurse. Of all the groups

of medical men, surgeons are the most dependent upon the nurse, and it, therefore, behooves us to take an active part in the determination of what should be the education of the nurse.

Very important changes are being made in the basic education of the nurse, many of which are excellent and many are not. The great danger to nursing as a profession today, in my opinion, is that its direction and control are largely in the hands of women of great crusading zeal whose minds are filled with fine theories but who often do not possess the practical knowledge and skill required for the training of efficient nurses. The possession of a so-called degree in nursing is certainly no guarantee of, or substitute for, a practical knowledge of nursing.

Many of the theories and some of the practices of the higher education for nurses have much to commend them, but these theories and practices need careful study and criticism and more than all else they need the constant direction of the medical mind and especially the mind of the surgeon.

In general, it is assumed that the nurse works under the direction of the medical man who is at least supposed to know something about her function and should have an active participation in her education, which he certainly does not have at this time.

Admirable as may be the efforts leading toward a higher education for nurses, what we surgeons, as well as medical men in general, need is women well trained to give adequate nursing care to the patient, and it certainly does not require four or five years to give such training.

It has been my observation and experience that the quest for so-called degrees in nursing does not improve the quality of the service rendered but tends to make the possessor of such a degree less efficient in the duties of the trained nurse and less qualified to impart the practical knowledge of nursing to her pupils. Her thoughts are too much occupied with educational themes and to little with the needs and care of the sick.

Unless we are on our guard, changes will be made in the education and practice of the nursing profession which will certainly not be helpful to the patient or the physician and of very questionable value to the nurse. The great danger is that the nursing mind, and the lay mind uncontrolled by the medical mind, will direct nursing education, with the result that the surgeon will have to take such nursing practice as is given him and try to like it. It is, therefore, very important that we surgeons take more active interest in the direction and control of nursing education and management in the hospitals with which we are connected.

Hospitals and their management are primarily and essentially the problem of the medical man and we should gladly accept this responsibility. There is no reason why the medical man should have a single track mind, and every reason why his mind should broaden to grasp and solve the professional problems having to do with the institutions in which most of his life is spent and most of his activities employed.

As professional men we should make every effort to preserve our liberty

of speech, thought and action and to resist the tendency to an abridgment of these liberties, no matter from what source it may come. This can best be accomplished by taking an ever active and unselfish interest in the management of the professional affairs of the hospitals and teaching institutions with which we are associated. Unless we exert ourselves, we shall certainly find the control and direction of our hospitals passing more and more into the hands of others less well qualified.

Enough has been said and written by administrative officials in Washington to indicate that their thoughts and plans are focusing on national regimentation of medicine. In peace as in war it is an old axiom that a sound offense is the best defense. In hospital affairs, as in national affairs, we should ever strive to be a self-governing people rather than a governed people.

The leaders of American surgery have for a long time believed that there should be some more distinguishing evidence of the ability to practice surgery than the mere possession of a medical degree. It was also fully realized that such standards of the ability to practice surgery should be determined by the profession and not by state or national legislatures. It was the crystallization of this sentiment which led to the formation of the American College of Surgeons some 25 years ago. It was believed that adequate training of the surgeon was an absolute necessity, but it was also appreciated that proper opportunities for such training must be provided. This resulted in an effort to survey and standardize the hospitals of the country, coupled with an untiring effort to raise hospital standards in the interest of the patient and also in order that better facilities for the training of the surgeon might be provided.

For many years inspection and grading of hospitals has been carried on more or less independently by the American College of Surgeons and the American Medical Association. This inspection has brought about a marked improvement in hospital standards and hospital service.

The Hospital Associations are also very much interested in this work and have given valuable assistance. There would appear, however, to have been a duplication of effort in these activities and it would certainly be of great advantage if all these activities could be combined under the cooperative guidance of one group composed of representatives of the most important associations concerned. This group in its activities and accomplishments could do for the elevation of hospital standards the same kind of effective work that the Council on Medical Education of the American Medical Association, the Association of American Medical Colleges and the State Medical Boards have done for the standards of medical colleges. Both the American College of Surgeons and the American Medical Association have in existence, and in function, not only most of the set-up required but also the nucleus of the personnel.

I think there will be general agreement that at the present time the surveys of hospitals are far too cursory and incomplete to allow of adequate and accurate grading. To properly survey a hospital requires qualities that are

acquired only by experience, and great care should be taken in the development of an efficient personnel. Such ability should command adequate salaries so that the turnover of the personnel would be as limited as possible. Such investigators of experience would be of the greatest assistance to hospital managements in advising wherein lay their defects and how best to correct them.

Inspection of all hospitals should be made at regular, and not too infrequent, intervals and gradings rearranged as conditions determined. The whole system of hospital inspection and grading needs revision and amplification; in fact this would appear to be one of, if not the most, urgent need of the hospitals of this country.

Unless some such effective organization as this can be brought into existence to function fearlessly, the problem as to where the surgeon is to be trained cannot be properly solved; and the effort to improve the training of the surgeon and to give him the mark of distinction conferred by the American Board of Surgery will fall far short of our anticipation.

To anyone familiar with the subject it is very evident that there is a complete lack of any basic standard for graduate training in surgery and it is equally evident that there is an imperative need for the formulation and adoption of some such basic standard as soon as practicable.

We must not only have more hospitals in this country so organized that the necessary training of the surgeon can be given, but there must be some way of knowing just what and where these hospitals are, so that the young man desirous of securing such training will know where to go.

While these remarks have been directed particularly to the problems of the surgeon, they are also in a great measure the problem of all branches of medicine, and it would seem reasonable to expect that some such cooperation as suggested would receive the general approval and support of the medical profession.

As a result of a careful study of the present incoordinated and overlapping activities of different groups, the suggestion has been made by Dr. Willard Rappleye, Dean of the School of Medicine of Columbia University, that a National Council on Medical Education be created, to be composed of representatives of the universities, medical schools, hospitals, practicing profession, specialty boards, state licensing bodies and public health agencies.

The functions of this proposed National Council on Medical Education would be those of studying the major educational needs of American medicine and of formulating adequate standards for these activities. Such a National Council could be of the greatest assistance in advising and directing the many agencies having to do with the health program of the nation.

Under the direction of such a National Council the problem of hospital grading and standardization could and would be more effectively solved since the organizations already having that important matter in their hands would have a better understanding of the real educational needs in each specialty field.

The creation of this Council must appeal to everyone as being the most practicable solution yet proposed for this intricate problem.

In brief summary an effort has been made to emphasize:

(1) The need of immediate efforts to provide adequate facilities for the training of the surgeon.

(2) More active, unselfish interest on the part of surgeons in directing the professional affairs of the hospitals with which they may be associated.

(3) The establishment of harmonious relationships between the professional group and the lay group in directing hospital activities.

(4) The development of a teaching atmosphere in hospitals.

(5) The need for and importance of efficiency and economy in hospital activities.

(6) The careful supervision and direction of nursing education by the medical profession.

(7) The imperative need of more careful inspection and accurate grading of hospitals.

It is quite beyond the scope of such an address as this to attempt to solve the problems which have been presented. All that can be done is to direct attention to them in the hope that the recognition of their importance and implications will be an aid in their solution.

THE NEED OF A NATIONAL COUNCIL ON MEDICAL EDUCATION, LICENSURE, AND HOSPITALS

WILLARD C. RAPPLEYE, M.D.

NEW YORK, N. Y.

DEAN OF THE FACULTY OF MEDICINE, COLUMBIA UNIVERSITY, NEW YORK, N. Y.

IT is a great privilege to be permitted to present to you certain considerations bearing upon medical training and practice. Probably at no time in our history have there been greater opportunities for leadership and guidance of public policies relating to medical questions. It is generally agreed that the essential feature of an adequate program of health services is a sufficient number of competent physicians. Upon medical education broadly conceived rests the responsibility of recruiting and training such personnel and of providing opportunities for practitioners to keep abreast of new knowledge and methods of diagnosis, treatment, and prevention.

A large number of separate organizations are dealing with different features of the whole problem, some of them with conspicuous success in their own spheres of influence, although a great deal of duplication, overlapping, competition, and confusion exists. At the same time there are important needs that no individual agency is covering satisfactorily and for which no organization feels responsible. It is of great importance that a thoughtful appraisal be made of our present efforts to meet the current needs and the impending demands upon the profession, and that we be prepared at least to consider ways and means of adapting our existing programs to meet more effectively the responsibilities which are likely to be placed upon us in the future.

Because of the special conditions in the early days of American medicine, the three functions of medical training, practice, and licensure were vested in the practicing profession. This was in contrast with the situation even at that time in most of the continental countries of Europe where for centuries medical education was the responsibility of the universities and licensure to practice was a function of the state. In Great Britain and France professional training was largely developed in the hospitals, with licensure resting in the agencies of the state. It is true that in this country there were some medical schools of high standing but most practitioners of 100 years ago had been trained by the apprenticeship method.

The historical background and tradition in this country of the control of medical training by the profession explains some of the present confusion and difficulties. Medical training now, however, has become the responsibility of medical schools, which are usually associated with universities. All students today must have a period of college preparation preceding the professional training; nearly every graduate takes a hospital internship; every state has its own agency for licensure; the new nationwide plans for graduate and postgraduate education involving the cooperation of the medical schools,

hospitals, and the profession have been instituted. These developments have occurred in segments and sometimes without much relationship to other parts of the whole structure of medical education or to the programs of other agencies carrying out parallel or duplicating activities.

Only a few items of recent history need be mentioned to suggest the various directions and many sources from which contributions have been made to our present standards of medical training. Just before the Civil War, most states by legislative action had removed the function of licensure from medical societies and had placed it under state boards of examiners. Much of this legislation was repealed at the time of the War. About 1870, agitation was renewed for the licensing of physicians by the state, largely because of the recognition of the dependence of sound clinical medicine and practice upon the discoveries in bacteriology, pathology, physiology, and the other sciences. By 1895, practically every state had created some kind of legislative organization regulating medical training and the examination and licensing of doctors. The legal enforcement by the different states of proper standards of training for licensure to practice has been one of the most important factors in elevating medical education in this country.

During the period mentioned above other important developments had occurred. The Association of American Medical Colleges was organized in 1891 to coordinate the educational efforts of the stronger medical schools. In response to the rapidly growing scientific content of medical training, Harvard University, in 1892, increased the length of the course to four years. A number of other schools promptly adopted similar programs. The founding of the medical school of Johns Hopkins University, in 1893, further stimulated the awakening interest in medical education.

In 1899, the graded curriculum for medical instruction was adopted generally in this country. At about that time the American Medical Association began its important work of collecting and publishing statistics on the medical school situation. In 1904, it created the Council on Medical Education which is responsible to the House of Delegates elected by the state medical societies. In 1909, that Council adopted as its standard the four year course which was in force in most of the leading schools at that time. Seventeen medical schools had already established the requirement of two or more years of college work for admission and 11 more made that regulation effective in 1910. This requirement was embodied in the minimum standard of the Council eight years later, at which time 81 of the 90 schools then in existence had adopted that requirement. It happens that eight states do not yet officially require two years of premedical college preparation although most of these states admit to their licensing examinations only graduates of approved medical schools. The monumental study by Mr. Abraham Flexner for the Carnegie Foundation for the Advancement of Teaching was published in 1910. That study and the publicity it received gave great impetus to the efforts to establish high standards of training and stimulated the needed financial support for medical education and research and for teaching hospitals.

Full credit is due to all the different individuals and organizations which contributed to the rapid elevation of the standards of medical training in this country, particularly to the courage, leadership, and financial aid of the universities and educational foundations, the participation of hospitals in the teaching plans, and the enforcement of standards by the state boards. There is no need of reciting here the contributions of the American Surgical Association and of the American College of Surgeons to the training and practice in surgery and the striking influence you have had on hospital standards in this country. The Association of American Medical Colleges and the universities have devoted great energy to the improvement of the basic undergraduate course. The Federation of State Medical Boards, individual state boards, and the National Board of Medical Examiners have rendered invaluable aid in their respective fields of action. The hospital associations are assisting in every way and now have greatly enlarged problems because of the newer demands upon them for better intern training and for graduate programs. All of you are familiar with the plans of the 12 American boards and of the Advisory Board for Medical Specialties which are largely responsible for the rapid and sound progress in graduate training. Special credit should be given to the Council on Medical Education and Hospitals, particularly in dealing with the proprietary, commercial, and weaker schools and in the collection and distribution of data on students, schools, state board activities, and other features of the whole program. While rules, regulations, and minimum standards have played an important part in the evolution of the present programs, the great strides have been made at levels well above the minimum standards by individual schools and universities under local leadership and by the desire of other institutions to emulate their successful undertakings.

As a result of the activities described and the increasing necessity of medical schools to provide adequate training in the medical sciences, which could not be met either by the weaker schools or by the commercial and proprietary institutions, the number of medical schools in the United States was reduced from 154 four year medical schools, in 1906, to the present 67. The graduates dropped from 5,364, in 1906, to 2,520, in 1922, but there has been a marked increase since that year. The number reached 5,377 in 1937, a total almost identical with that of 1906. In other words we are today graduating as many physicians from 67 medical schools as we did from 154 institutions 32 years ago. That a number of schools have enrolled more students than they can educate in keeping with present day standards has been recognized for years. During the last three years, however, the entering classes of certain of the schools have been reduced. In time the size of the student body in some of the institutions will be better adapted to their educational facilities and teaching programs. The figures cited do not include the additions to our profession annually from Canadian and foreign sources, from unapproved institutions, and from Americans who study abroad.

It is common knowledge that, despite efforts of the last 30 years to standardize medical education, "wide differences continue to exist in buildings,

equipment, personnel, students, financial support, hospital facilities, and educational policies." In response to this situation a reinspection of the medical schools made recently by the Council on Medical Education has shown that about 20 of the institutions approved by that Council do not even now provide a fully satisfactory preparation. If it is true, as stated by the Commission on Medical Education, that "an emphasis on educational principles in medical training and licensure can be secured only by modifying the point of view and broadening the interests of those responsible for medical education and licensure, not by recommendations, statistics, new regulations, further legislation, or manipulation of the curriculum," the evaluation of medical school objectives and programs can best be secured not by an agency representing the profession alone, which really is the alumni body of the schools, but by one which represents fully as much the educational, hospital, licensing, and other phases of this problem.

Students entering medicine prepare in about 600 colleges and universities. The requirements for admission vary considerably. There is a wide range of opinion on the objectives and content of preprofessional education. The basis of selection by different schools is not only undefined but frequently contradictory. Recent developments in graduate fields of instruction emphasize the need of better criteria of selection at the source. The situation is confusing to students and to those responsible for the conduct of the colleges and universities. There are numerous problems relating to general and medical education pressing for study and solution, yet there is no convenient mechanism in existence by which these mutual problems of medical schools and colleges can be discussed and defined.

The increasing dependence of sound medical education upon individualized, supervised experience in the teaching wards and clinics by means of the clinical clerkship presents special problems for the hospitals. The internship has become universally recognized as an essential part of the basic preparation for practice. Twenty states now require such a training for admission to the licensing examinations. Certain of them so define and regulate this period of training, however, that they defeat the efforts of universities, medical schools, and hospitals to provide a satisfactory preparation adapted to the needs of those going into different fields of practice. Uniformity, rigidity, and regulation are not distinguishing characteristics of an educational program. It is well known that the intern period is poorly adjusted in many hospitals to the preceding medical course, to the needs of the student, and to subsequent graduate training. Even the approval of national evaluating bodies is uncertain. An intensive study of internships in a group of eastern hospitals recently has shown that not more than a half of those hospitals approved for intern training by the Council on Medical Education and Hospitals provide satisfactory educational standards. The proportion of residencies meeting a real educational level is smaller. There is need for joint and continuing study of the place and functions of the internship and

residency in the evolution of the medical course, graduate training, and licensure.

Excellent cooperation exists between most of the state medical boards and the medical schools and between the large majority of the different states on matters of reciprocity and indorsement of educational credentials. The National Board of Medical Examiners, organized in 1915, has been very helpful in establishing a national point of view regarding licensure. Many believe that medical licensure in the country as a whole could be simplified through some joint action by the Federation of State Medical Boards, the Association of American Medical Colleges, and the National Board of Medical Examiners. It is reasonable to assume that eventually this problem will be dealt with in a manner analogous to that found satisfactory in other countries. At the present time no agency exists for study and integration of this important public and educational function.

Plans already developed for graduate and postgraduate training will require wide and, in some instances, fundamental readjustments in hospital services, if the hospitals are to participate fully in these newer opportunities. The medical schools and universities are being called upon to assume responsibilities in these same programs. All are being subjected to numerous surveys and inspections by different agencies, frequently overlapping in their interests and conflicting in their objectives. The 12 American specialty boards and the Advisory Board for Medical Specialties, created in 1933, recognize the dangers of rigidity, regimentation, and regulation in the field of graduate training which must depend so largely upon the educational initiative, self-reliance, and resourcefulness of the individual. The upward extension of medical education into the graduate fields should be based, with necessary adaptations, on those principles of selection of students, concepts of learning, forms of instruction, and other features which characterize true graduate education.

Sound plans for the evaluation and approval of graduate programs cannot be evolved by a single agency but call for cooperative action by a group representing the various major interests involved. The proposals, for example, that national and state registers or directories of specialists be created and that general practitioners be certified for continuation instruction have definite relationships to present methods of licensure. The state agencies should obviously be brought fully into the general plan.

The premedical student, the medical student, the intern, the hospital resident, the general practitioner, the specialist, and the public health administrator should be regarded from an educational point of view merely as different phases of the training of personnel to meet the health needs of the country. The problems from college preparation to retirement from professional life should be looked upon as parts of a single educational program. Portions of the program are primarily within the jurisdiction of universities, some are largely within the domain of the hospitals, others are in the various fields of practice, and some are under governmental regulation.

It is becoming increasingly apparent to those familiar with the situation that there is need of coordination of the various phases of medical education and better definition of the several areas of responsibility of national and state agencies, universities, hospitals, and professional bodies dealing with portions of the whole program, if medicine in this country is to meet fully its obligations. Reluctant as one may be to see another agency in medicine, the logical conclusion from the present more or less unrelated and frequently overlapping efforts is to create a national coordinating body representative of the major activities in medical education and service in order more effectively to meet the new conditions and needs of the country.

A National Council on Medical Education, Licensure, and Hospitals should be created from within our present organizations, made up of representatives of the universities, medical schools, hospitals, practicing profession, specialty boards, state licensing bodies, and public health agencies. There should be no difficulty in securing full representation of leaders in every subdivision of medical education and practice, hospital activities, licensure, and public health on such a central body. If such an organization is created the modest financial support from voluntary sources should not be difficult to obtain.

The functions of the proposed National Council on Medical Education, Licensure, and Hospitals would be those of studying the major educational needs of American medicine, of mobilizing the best current opinions regarding the different phases of professional training at its several levels, of formulating adequate standards for these activities, and of advising regulatory bodies and governmental agencies on standards, methods, procedures, and areas of action. The National Council should, among other things, delegate to existing organizations all administrative functions and endeavor to coordinate the efforts and simplify the procedures of the multiple agencies now in operation. A central clearing house carrying influence and prestige by virtue of the knowledge and judgment of its personnel and providing a suitable vehicle of our own creation for cooperation on matters dealing with all features of medical education, transcending the activities and interests of any single group or organization, would be of the greatest practical value to the profession, the universities, the hospitals, the licensing bodies, and the future health program of the entire country.

DISCUSSION.—DR. EVARTS A. GRAHAM (St. Louis, Mo.): I think that Doctor Rappleye has unquestionably put his finger directly on a great need which exists in this country and which has existed for a long time.

Some of you may think that the functions of this proposed Council are already being carried out to a considerable extent by existing organizations, as, for example, the Council on Medical Education of the American Medical Association. That is hardly correct, however, because the Council on Medical Education of the American Medical Association, for instance, is a body which really represents only the American Medical Association. It is not representative, at least so far as appointments are concerned, of the medical schools of the country, the universities and the various other elements which Doctor Rappleye mentioned in his paper.

Despite the fact that, as has been mentioned by the President in his address

and has been intimidated by Doctor Rappleye in his paper, we are living in a bureaucratic age, despite that fact, it does seem as if it would be desirable and necessary to have another council set up which, however, I would infer from Doctor Rappleye's paper, would not have any direct authority allocated to it except an authority of prestige. Perhaps he will make this point a little more clear in his closing remarks.

Certainly there is a danger in too much standardization of education in any form. There would be a danger in setting up a body which would have a thorough standardization; that is to say, a legal authority, perhaps, to tell the medical schools exactly what they should do and exactly what they should not do. This probably would be destructive of the very principles of sound education.

We have seen in recent years in Europe too much of this tendency to wish to welcome it here in the United States. I am quite sure, from talks with Doctor Rappleye about this matter, that he does not mean that this council which he proposes should have any such Fascistic power as some might fear for it.

There is no question about the fact that there is a need of a coordinating body to coordinate all of the activities and functions of the various independent bodies, which now exist in a large number in this country, all attempting to aim at the same goal, more or less, but missing that mark to a considerable extent because of duplication of effort, because of failure sometimes to grasp the essential point in the strategy of the whole campaign, and because too often the emphasis is placed on the details rather than on the fundamental principles involved.

Actually, of course, the most important and the most fundamental cog in the whole machine of the care of the sick is the doctor. One of the most important elements in the matter of how good a doctor is or how poor and ineffective he is, is the question of the training of that doctor, his education and the facilities which are offered to him to keep abreast of developments which have taken place in medicine since the time that he left the doors of his medical school.

Any plan of improvement of the care of the sick on a large scale which does not take into serious consideration, as one of the most fundamental steps necessary, the improvement of the educational facilities for the practicing doctor will, of course, fall far short of its goal.

We read in the newspapers a few months ago about a recommendation, for instance, which had been made to the national government that small hospitals—the newspaper account which I read stated hospitals of 30 to 60 beds—should be erected throughout the country in order that the isolated sick in the rural communities could be taken care of properly, rather than to undergo the hardships of being taken care of in poorly equipped homes.

It is easy, of course, for anyone who knows what the practice of medicine is all about to realize immediately that if the country should become studded with hospitals of 30 to 60 beds, serious effects would result, which would be, in the first place, that these hospitals would be in competition with the large private hospitals throughout the country, which have had a splendid record in unselfish training of the medical profession to go out and take care of the sick. It would mean that these small hospitals are totally inadequate themselves to provide proper facilities for the responsibility of training doctors. It would mean, therefore, that probably in general, the public as a whole would suffer from such a plan instead of being improved by it.

I take it that the function of such a council as Doctor Rappleye has in mind would call attention to such a serious defect, for example, in such a proposal;

and with the weight of prestige behind it, as containing representatives of various important bodies, would perhaps have enough influence to curtail efforts which might be made from time to time by well meaning individuals to propose schemes for the welfare of the sick which actually would not be practical; and would present such defects as those I have just mentioned in regard to this particular instance.

There is really nothing new about Doctor Rappleye's proposal, as he has indicated. It happens merely that we in the United States have been slow to put such a measure into operation. I believe I am correct in saying that the British Medical Council, which has been in existence for nearly 100 years, has been and is an organization which carries on many of the functions which Doctor Rappleye proposes, for example, for this Council, the formation of which he advocates. Perhaps he will elaborate on that a little more in closing his discussion.

Finally, Mr. President, I should like to ask Doctor Rappleye what he wishes to do about this. I am not quite sure whether he wishes a resolution of some kind from this Association, expressing approval of the creation of such a National Council, or whether he merely wishes to think about this matter for a while and perhaps some time later discuss it again. I should like, Mr. President, to ask you to ask him, if you will, whether he wishes the American Surgical Association to do something about it.

DR. HAROLD L. RYPINS (New York State Board of Medical Examiners): I think the best light I can throw on Doctor Rappleye's very important contribution is to draw attention to the fact that within the last 10 or 15 years, there have been at least half a dozen sporadic movements in exactly the direction that he is pointing out now, and that all of these movements have come about through the necessity for the expression of opinion and sometimes of action from various representatives of all interested parties in medicine.

For example, all of you are familiar with the fact that the National Board of Medical Examiners, which has been functioning now, I think, for about 20 years, has representatives in practically every interested medical organization throughout the country. It has just dawned upon me what a monumental piece of politics and strategy that Board has accomplished in actually taking over some of the functions of 40 odd governmental agencies without the least amount of bad feeling.

It has accomplished something which none of the individual boards could ever have accomplished, and it has done so by taking some of the functions of those boards away, and still there has been absolutely no friction between the state boards, which are governmental agencies, whose members are mostly appointed by governors, and the National Board.

The answer is this: Apart from the very great political skill of Doctor Rodman and his associates, the Board has had such a broad representation in its make-up that there could be no question of its prestige and of its public interest. The fact that it has been able to accomplish what it has is a very good indication of what a National Council, such as suggested by Doctor Rappleye, might do without any friction whatsoever.

About 10 years ago, it became apparent that we ought to know something more about medical education; therefore, a Commission on Medical Education was formed. This, again, was made up of representatives of practically every organization in the country interested in medical education. No administrative function was assumed in this case. Simply, a study was made and a lot of facts were collected. There was some deliberation upon these facts and a report was finally made which contained very vague, if any, recommenda-

tions. Nevertheless, the effect of this Commission was exceedingly far-reaching upon the advance in medical education in this country. In fact, it was more far-reaching than any more specific regulations and laws that had been heretofore laid down as to how to conduct medical schools.

As far as I know, there was no serious friction between the Commission on Medical Education and the various interested bodies.

We are at the present time in the throes of organizing a Commission on Graduate Medical Education, which we hope will operate in exactly the way that the earlier Commission did, and which will bring together some information and some ideas about graduate medical education which none of the present existing agencies seem to have been able to bring to the attention of the public.

Again, about seven or eight years ago, a very serious problem arose, when it was suddenly discovered that a very large number of American boys who were able to gain admission to foreign medical schools were studying in Europe with the expectation of returning to this country to practice. This problem was brought to the attention of the various individual bodies concerned from time to time, but no one of them seemed able to cope with the problem. Finally, the numbers increased to something over 2,000 a year, and the situation became so acute that it became necessary to form a Joint Committee on Foreign Medical Students which could represent the interests and speak for all the various bodies concerned.

As soon as we had one committee which had authority to speak for everyone in American medicine and American licensure and American hospitalization, we were able to deal very adequately, and simply, with the representatives of the European governments, but no one single agency would have been able to do so.

Then again, during the last 15 years, at least, there have been developing, as you know, the various boards for medical specialties, but in spite of the fact that several of them were very well organized, no one seemed to be able to work out a program which would smooth over the difficulties between the various boards—the difficulties arising between those boards and the organized medical profession.

About four, certainly not more than five years ago, it was deemed advisable to create an Advisory Board of Medical Specialties which was to be made up of representatives of all the interested bodies, and which was to interject itself between the medical profession as organized and these individual bodies as organized. The result of this interjection by this broadly organized group has been the complete organization and, as you know, operation of the 12 specialty boards which are deemed to be desirable at the present time.

I think there is no question that had there been no advisory board which could speak for all the interested bodies, there would still be an amount of unnecessary jangling between the individual boards and between the individual boards and organized medicine as a whole.

I bring these facts to your attention. I might also mention the survey of medical schools which Doctor Rappleye spoke of and in which there was some cooperation between the Council, the Medical Schools Association and the Federation of State Boards.

I bring these six points to your attention (and I believe many more could be recited) to show that during the last 15 or 20 years, the need has arisen for some sort of National Council with a broad representative base, and the prestige that comes from such representation, to meet the problems of mutual

interest that are always developing. Within the last 15 to 20 years, we have had at least six such occasions.

Now, if the government in Washington is going to continue as actively as I believe it is, I believe seven or ten occasions will arise very shortly; and I agree with Doctor Rappleye that it is very much in the interest of the medical profession, and even more in the interest of the public health, that there be a genuinely representative organization that can speak with intelligence and authority for the entire medical profession and the lesser individual bodies, whose important functions should certainly not be interfered with.

DR. WILLARD C. RAPPLEYE (closing): Doctor Graham brought up the question of authority, and he is quite right in interpreting what is certainly in my own mind, and I think in the minds of most of the persons who have been thinking about this problem and seeing the situation develop, that we are concerned primarily with getting together a group of persons who will represent the leadership of ideas and carry with it the prestige and weight of authority that would arise by having real judgment on a great many of these interlocking problems.

We are all prone, of course, to think of our own situations and problems, and I think we need some agency that is extending over and above the interests and responsibilities of single agencies as we now have them, many of which are doing excellent work in their respective fields. The whole idea back of the proposal is to make progress at the top as well as at the bottom. Leadership is going to be always at the top.

There is no question, and the fact remains obvious to everyone, that the government is involved in the program of medicine and is going to become a great deal more so. It seems to many of us that we ought to become organized in advance and be prepared to give that authoritative advice and counsel that it ought to have. I am not sure that they will take it all the time; we don't expect that, but certainly we have to be in a position, at least, to have some mechanism set up within the profession to help in guiding many of these problems that cut across all of our national agencies, hospitals, medical profession, licensing bodies and similar activities.

The question of the General Medical Council: Those of us who have been interested in the history of that organization, and other plans of medical education and licensure in other countries, have been very much struck with the fact that the situation in this country, at the moment, is almost identical with that leading up to the organization of the General Medical Council, in 1858. We have similar problems. Many of the situations that we have today are closely paralleled with those that led to the formation of the General Medical Council.

I would like to speak about what Doctor Rypins has said. He has brought out the very point that this technic of cooperation between the agencies is going on and there are a number of activities already set up, many of them temporary, that have gone forward with the idea of formulating joint programs that have a bearing on medical training.

This is not a radical suggestion. It was embodied in the report of the Commission on Medical Education to which Doctor Rypins referred, which was printed in 1932. It is only following along what has been going on for the last 100 years in medical education in other countries, and in recent years in this country.

THE TREATMENT OF CRANIAL OSTEOMYELITIS AND BRAIN ABSCESS

ALFRED W. ADSON, M.D.

SECTION ON NEUROLOGIC SURGERY, THE MAYO CLINIC

ROCHESTER, MINN.

SUPPURATIVE lesions of the scalp, skull, accessory sinuses, meninges and brain bear an intimate relationship to one another since the infection may spread by contiguity or travel along lymph and diploic channels, and along emissary veins which communicate with veins that pass through the dura into the substance of the brain. Appropriate surgical treatment of the localized infection often will limit its extension and prevent the more serious involvement of the meninges and brain.

The treatment of osteomyelitis of the skull does not differ from the treatment of osteomyelitis of other bones; removal of all necrotic and infected osseous tissue is required in addition to sequestrectomy. However, special consideration concerning these operative procedures is necessary, since the scalp and periosteum may have been destroyed, and removal of infected bony tissue may result in exposure of the meninges; moreover, brain abscesses are frequent sequelae of osteomyelitis of the skull.

A consideration of the anatomic arrangement of the veins in the diploe and of the emissary veins is in order, so that it may better be understood how infections may travel through communicating veins to give rise to distant areas of osteomyelitis, with or without accompanying brain abscess. Infective emboli in the arterial system are rarely responsible for osteomyelitis of the skull. Thrombosis of vessels in an extending infection of the scalp frequently spreads the osteomyelitis. The arterial supply to soft tissues and haversian canals about an osteomyelitic region serves as a barrier to limit the progress of infection by maintaining the life of the osseous tissue. Frequently, the osteomyelitic process will destroy but one table of the skull when the circulation to the opposite table has been maintained.

Gray²² states: "The diploic spaces of the cranial bones in the adult contain a number of tortuous canals, the diploic canals (*canales diploici* [Brescheti]), which are surrounded by a more or less complete layer of osseous tissue. The veins they contain are large and capacious, their walls being thin and formed only of endothelium resting on a layer of elastic tissue; they present at irregular intervals pouch-like dilatations, or culs-de-sac, which serve as reservoirs for the blood.

"In adult life, so long as the cranial bones are distinct and separable, these veins are confined to the particular bones; but in old age, when the sutures are united, they communicate with one another and increase in size. They communicate, in the interior of the cranium, with the veins and sinuses of the dura, and on the exterior of the skull with the veins of the pericranium. They

consist of: (1) The frontal diploic vein (*v. diploica frontalis*), which opens into the supraorbital veins by an aperture in the supraorbital notch; (2) the anterior temporal diploic vein (*v. diploica temporalis anterior*), which is confined chiefly to the frontal bone, and opens into one of the deep temporal veins through an aperture in the greater wing of the sphenoid; (3) the posterior temporal vein (*v. diploica temporalis posterior*), which is situated in the parietal bone, and terminates in the lateral sinus through an aperture at the posteroinferior angle of the parietal bone or through the mastoid foramen; and (4) the occipital diploic vein (*v. diploica occipitalis*), the largest of the four, which is confined to the occipital bone, and opens into the lateral sinus or the torcular Herophili.

"The emissary veins (*v. emissaria*) are vessels which pass through apertures in the cranial wall and establish communications between the sinuses inside the skull and the diploic veins in the diploe, and the veins external to the skull. Some of these are always present, others only occasionally so. They vary much in size in different individuals. The principal emissary veins are the following: (1) A vein (*v. emissarium mastoideum*), almost always present, runs through the mastoid foramen and connects the lateral sinus with the posterior auricular or with the occipital vein. (2) A vein (*v. emissarium parietale*) which passes through the parietal foramen and connects the superior sagittal sinus with the veins of the scalp. (3) A plexus of minute veins (*v. rete canalis hypoglossi*) which pass through the anterior condylar (hypoglossal) foramen and connect the occipital sinus with the vertebral vein and deep veins in the neck. (4) An inconstant vein (*v. emissarium condyloideum*) which passes through the posterior condylar foramen and connects the lateral sinus with the deep veins of the neck. (5) A plexus of veins (*v. rete foraminis ovalis*) connects the cavernous sinus with the pterygoid and pharyngeal plexuses through the foramen ovale. (6) Two or three small veins run through the foramen lacerum medium and connect the cavernous sinus with the pterygoid and pharyngeal plexuses. (7) There is sometimes a small vein connecting the same parts and passing through the inconstant foramen of Vesalius, opposite the root of the pterygoid process of the sphenoid bone. (8) A plexus of veins (*plexus venosus caroticus internus*) traverses the carotid canal and connects the cavernous sinus with the internal jugular vein. (9) A small vein (*v. emissarium occipitale*) usually connects the occipital vein with the lateral sinus or the torcular Herophili and the occipital diploic vein. (10) A vein is usually transmitted through the foramen cecum and connects the superior sagittal sinus with the veins of the mucous membrane of the nose."

OSTEOMYELITIS.—*Etiology:* Osteomyelitis results from infection of an avascularized bone or from extension of an infection into the diploic canals.¹⁵ The most common sources for the infection are contaminated, compound, comminuted fractures of the skull, furunculosis of the scalp and extension of infections from the ear and accessory nasal sinuses, the frontal sinus being the chief offender. Diffuse osteomyelitis of the outer table is more prone to

afflict children and young adults than is osteomyelitis of the inner table of the skull, since the outer table is more porous. *Staphylococcus pyogenes aureus* is chiefly responsible for osteomyelitis resulting from infections of the frontal sinus and is the organism which frequently produces the osteomyelitic processes in compound comminuted fractures, with infected, lacerated wounds.

The denuded skull, resulting from burns by electricity and fire, from extensive lacerations, and from removal of the scalp because of tumors, ultimately, undergoes destructive processes. If the circulation to the inner table is intact, only the denuded outer table will degenerate and separate as a sequestrum.³³

Craniotabes, gummatous osteomyelitis,¹ caused by syphilis, is a representative of the group of chronic infections. *Eberthella typhi* (*Bacillus typhosus*) at one time was a fairly common cause of osteomyelitis but today, since the introduction of vaccination, is rarely seen. An occasional case results from tuberculous involvement of the skull. Actinomycosis, too, occasionally is responsible for infective processes of the skull.

Pathology.—The process of osteomyelitis begins with the introduction of suppurative organisms into the vascular channels of the skull. If the bone is denuded of its scalp, the process will extend until adequate circulation is encountered. Frequently, accompanying cellulitis will destroy the circulation of the scalp about the denuded bony area and this further encourages extension of the osteomyelitis. The serpiginous extension along diploic canals will give rise to additional areas of necrosis beyond the original focus.³⁵ The infection of diploic veins results in thrombosis and the development of granulation tissue. This process may extend either to the pericranial or to the intracranial structures by extension along emissary veins. Phlegmons of the scalp develop over the necrotic bony areas and extend the infection over suture lines to new areas, through emissary and diploic veins, thus giving rise to additional osteomyelitic processes.²⁸ The disease may progress until the entire skull has been involved. On examination of the skull, islands of normal bone will be found between necrotic and sequestered areas. These islands apparently have resisted the infection, owing to the fact that the circulation has been maintained and that a zone of granulation has served as a barrier in limiting the infection in the diploic canals.

The reparative process of granulation and absorption works in conjunction with the destructive process. As the bony cells die they disintegrate, are absorbed and are replaced by granulation tissue. This is readily demonstrated in a denuded area of skull. The exposed bone at first appears dry and dead-white, but sooner or later a zone, or ring, of granulation will appear at the scalp margin. In the zone of granulation there will develop a localized osteomyelitis which eventually destroys a ring of bone in the outer table of the skull. When this has taken place, the granulation tissue in the diploic spaces will actually lift from the dead-white outer table of the denuded skull (Fig. 1). Removal of the granulation tissue will reveal that the inner table is usually intact and is very vascular, owing to the fact that its circulation has been main-

tained by small arterioles from meningeal arteries. However, if the scalp has been destroyed by a severe burn, both tables of the skull will slough away spontaneously.

Symptoms.—The local symptoms are usually preceded by a history of infection. If the symptoms follow injuries of the scalp and skull, a septic temperature of low grade develops, associated with leukocytosis and with localized swelling of a soft and doughy consistency, with or without localized tenderness. If the infection results from frontal sinusitis, the swelling will appear over the frontal bone, usually on the side of the involved sinus.⁷ When the infection is of hematogenous origin, as it is in syphilis, the swellings may

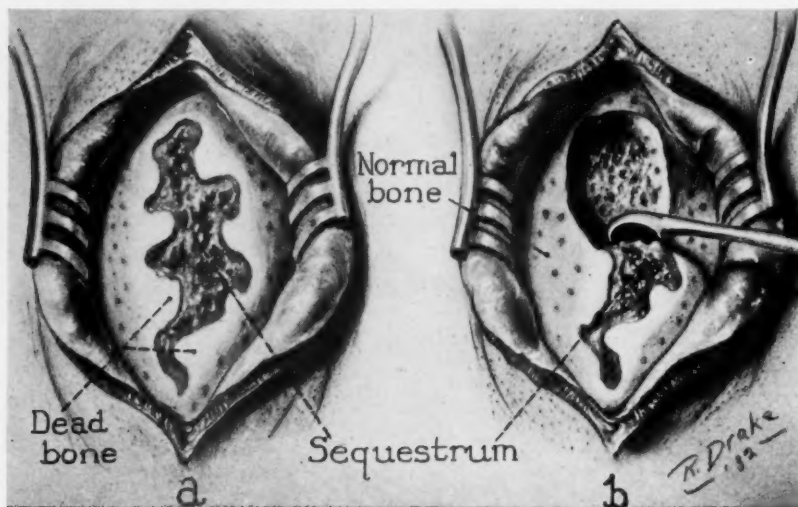


FIG. 1.—(A) Drawing showing worm-eaten erosions of osteomyelitis involving the outer table. (B) The procedure employed in removal of the sequestrum and the overhanging ledges of dead, white bone.

occur in numerous parts of the skull, but usually in those parts where the cranial tables are thickest, and where the diploic spaces are largest, as in the parasagittal portions of the frontal and parietal bones. Roentgenologic examination usually reveals moth-eaten erosions of one or both tables of the skull. All roentgenograms should be taken in two directions in order to demonstrate the extent of the lesions.

Surgical Treatment.—The phenomena of destruction and repair form a basis for surgical treatment, since adequate drainage of suppurative lesions of the scalp, removal of infected bony fragments and removal of dead and necrotic bone will aid in preventing or limiting the osteomyelitic process.⁴ Small puncture-like incisions over fluctuating areas are of some value but are not sufficient surgical procedures to check the process. Localized osteomyelitic areas will continue to spread until the scalp has been reflected and all of the dead and sequestered bone has been removed. Frequently the necrotic bone can be removed with a sharp curet or gouge without removing both tables of the skull (Figs. 2 and 3). However, there is less danger of the



FIG. 2.—(A) Two electric burns on the left parietal area which had been present for five months without evidence of granulation. (B) Spontaneous sequestrectomy aided by the use of a forceps, which permitted granulation and healing of the wound. (C) Photograph taken six weeks following the photographs (A) and (B).

infection extending into the brain with removal of both tables of the skull, if they are necrotic, than there is when the inner table is left in place and only the outer table is removed. The dura serves as an excellent barrier to inward extension of infection provided it is not injured in the course of sequestrectomy. If extensive areas have become involved, it is reasonably safe to uncover as much as one-fourth of the skull at a time. At each stage of the operation the removal of dead and necrotic bone should be complete; however, islands of normal bone, when present, should be left in place. The periosteum likewise should be preserved and resutured after the infectious material has been removed and the field cleansed with tincture of iodine. If drainage is instituted, the drain should be removed within 48 hours, for not infrequently primary healing will be obtained. The additional areas should be treated in a similar manner at intervals of four to five days between each two operations, until all of the necrotic bone has been removed. Too often

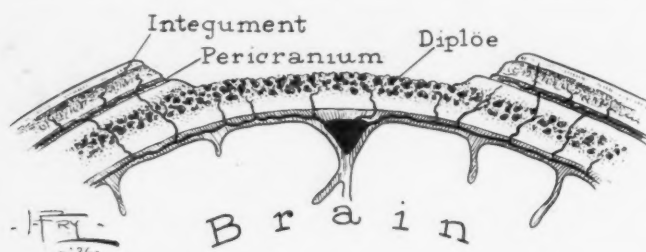


FIG. 3.—Drawing illustrating the importance of removing the necrotic outer table of the skull peripherally until normal bone is exposed, which also permits the edges of the skin to cover the fresh margins of the bone.

the surgeon is inclined to leave dead-white bone lateral to the osteomyelitic process. If this is done, the process will continue. The dead bone should be curetted away until bleeding appears from its cut edges (Figs. 2 and 3). The bleeding can be controlled with strips of gauze soaked in tincture of iodine which, if left in place, should be removed within 48 hours, together with other drainage material such as Penrose drains and rubber tubes, etc.

Epithelization of denuded areas of the skull can be materially hastened by removing the outer table in order to expose the diploic spaces. This will give rise to granulation tissue which serves as a bed for the epithelium (Fig. 3). Skin grafts can be employed to hasten the process of epithelization. The outer table is readily removed and the first step in the removal is to make multiple openings in the outer table with the trephine bur. After this, the ridges of bone are rongeuired away or removed with a chisel, care again being taken to remove all dead bone, even though it may extend under the margin of the scalp. The uncovered inner table, with its oozing diploic veins, is protected by perforated paraffin gauze, which also encourages epithelization.

Although radical surgical treatment is the effective means of controlling the osteomyelitic process, it should be borne in mind that the virulence of the organism, the resistance, and the specific immunity of the patient to the particular organism are the combative forces which determine the activity of the

process and the extent to which it will travel. This being true, active supportive measures should be employed. Administration of vaccines has been suggested and sulphanilamide has been employed, both of which have proved of value. High caloric diets with adequate vitamins are essential. Occasionally local application of heat has aided the circulation and hastened development in the zone of reaction, thus limiting the process.

The accompanying leptomeningitis is a serious complication and is controlled best by repeated or continuous spinal drainage and the administration of sulphanilamide. Again, a nourishing dietary regimen and excellent nursing are material aids in control of the disease.



FIG. 4.—Roentgenogram, lateral view, showing extensive osteomyelitis of the left temporal, parietal, and frontal bones.

The cerebral abscess which results from osteomyelitis is one of the suspected and serious complications. When it occurs, the surgeon is confronted with the problem of deciding whether to drain the abscess before treating the osteomyelitis or to treat the osteomyelitis before draining the abscess or, indeed, to attempt to treat both at the same time. A maxim of general surgery should be invoked in the treatment of these lesions, namely: "A patient often will survive two major operations if they are performed separately but may fail to survive if both are attempted at the same time." Too much never should be attempted at one operation. Judging from my own experience, it is better to delay drainage of an abscess than to delay the operative procedure for control of the osteomyelitis. Encapsulation becomes more nearly complete if drainage is moderately delayed, whereas, the osteomyelitis process will

continue to extend and may give rise to additional brain abscesses. A number of times I have removed the necrotic bone, resutured the scalp and drained the abscess a week later through a separate incision without lighting up the osteomyelitis (Figs. 4, 5, 6 and 7). There is an occasional exception, namely: when the process has continued for several weeks; when the cerebral symptoms produced by the abscess are very marked; and when the osteomyelitic process is limited and apparently controlled. In those instances the cerebral abscess has been drained first and the osteomyelitis treated following removal of the drains. There is also the occasional case in which the osteomyelitic area is very circumscribed, and the abscess appears to be situated close to



FIG. 5.—Roentgenogram, lateral view, made following removal of sequestrum and dead bone illustrated in Figure 4.

the surface. Then it is possible to drain the abscess and remove the necrotic bone at the same time through the osteomyelitic area. There is a constant danger of producing leptomenigitis in opening the dura in the presence of infected material. There is also the danger of producing an abscess by introducing a needle through infected dura into the brain when searching for a subcortical abscess.

BRAIN ABSCESS.—Etiology: Brain abscess results from invasion of the brain by pyogenic organisms from acute or chronic infection of the middle ear; frontal or accessory nasal sinuses; compound fractures and penetrating injuries of the skull; infections of the scalp or osteomyelitis, and from infected emboli arising from bronchiectasis, empyema, endocarditis, and general

CRANIAL OSTEOMYELITIS



FIG. 6.—Roentgenogram, anteroposterior view, following sequestrectomy and drainage of abscess of left temporal lobe; drainage tubes in place.



FIG. 7.—Photograph of patient following sequestrectomy and drainage of abscess of the left temporal lobe.

septicopyemia.^{5, 16} Although many abscesses result from an extension of the infection by contiguity, a large number develop as a result of retrograde infection of a thrombosed vein within the brain which communicates with veins that drain infected portions of the scalp, skull or meninges.¹⁰ Occasionally, a stalk or channel may be found to extend from the region of suppuration to the abscess but, more often than not, the cerebral abscess is situated apart from the primary focus.³² They are situated, usually, in the white matter of the brain where the veins originate and where the circulation is less abundant.

Each abscess passes through three stages of development: The initial stage is that of localized encephalitis which might undergo recovery spontaneously, producing a syndrome of pseudo-abscess. The second stage is that of liquefaction and necrosis with encapsulation. As immunity progresses, the general symptoms of infection subside. The abscess becomes quiescent except for the symptoms of intracranial pressure and localization. The third stage represents the terminal phase of activity. Small abscesses disappear spontaneously by inspissation or resolution of the pus. Large abscesses are very likely to rupture into the ventricle or subarachnoid space and produce death unless properly drained. Therefore, appropriate treatment requires proper medical management during the initial stage and the selection of a suitable operation at a time when drainage will be most effective during the second stage. The procedure should afford adequate drainage without spreading the infection or giving cause for recurrence of the same abscess.

Macewen,³¹ in 1893, was the first to recognize and localize a brain abscess but, unfortunately, he was not permitted to operate. However, he verified his diagnosis at necropsy. This experience prompted him and others to advise surgical treatment for cerebral abscesses. In reviewing Macewen's book, it is apparent that his good results were owing chiefly to the fact that most of the abscesses were encapsulated. The encapsulation suggests that the patients were brought to him for treatment after abscesses had passed through the initial stage into the second stage of development. At this time, the organisms are less virulent and many have died. The immunity, likewise, has reached its maximal efficiency.

Owing to Macewen's good results, many surgeons advise operation as soon as possible after a diagnosis of brain abscess has been made. However, they fail to recognize the fact that clinical diagnosis has improved since his time and that diagnosis of brain abscess now is frequently made in the initial stage instead of in the second stage. These early operations are responsible for the increased mortality. A suppurative process of the brain is not different from a suppurative process of any other part of the body. Therefore, similar reasoning and similar treatment should be employed. "Wait until the abscess is ripe before opening."

Infective Agent.—The organisms most frequently found in pus removed from cerebral abscesses are *Staphylococcus albus*, *Staphylococcus aureus*, pyogenic *Staphylococci*, and hemolytic *Streptococci*. Other varieties of bacteria

have been found, the nature of which depends on the source and character of the cerebral contamination.

Pathology.—Cerebral abscesses are invariably situated below the cortex.¹⁹ Occasionally, a stalk can be seen to extend from the meninges to the abscess, but more often than not, trace of path of the invasion cannot be demonstrated. The explanation for this, I believe, is that the communicating vein, which served as a path for the organism to invade the white, poorly vascularized brain tissue, has become thrombosed. The stalk, if present, represents the zone of reaction about the infected vein. Following the inoculation of the brain, a localized region of encephalitis results. This process extends by thrombosis of more capillaries and vessels until reaction and immunity limit such extension. The center of the lesion disintegrates, liquefies, and becomes pus. Proliferation in the peripheral zone results in formation of fibrous tissue and encapsulation.

I am convinced that infections may travel through thrombosed veins and diploe for long distances, to produce abscesses without osteomyelitis. This is substantiated by the fact that abscesses do result from infections of the scalp, frontal sinus, and antrum without an accompanying osteomyelitis. The reason cerebral abscesses do not always follow osteomyelitis of the flat bones of the calvarium, frontal sinusitis, disease of the middle ear, or mastoiditis, I believe, is that venous thrombosis is limited by a zone of reaction and a collateral venous circulation. Positive blood cultures are rarely obtained. Symptoms of meningitis frequently accompany cerebral abscesses.

A single embolic abscess is relatively rare. Multiple abscesses follow pulmonary disease, particularly bronchiectasis. Also, they may represent a part of a general septicopyemic infection. The virulence of organisms producing multiple abscesses is usually so overwhelming that death results before encapsulation takes place.

Penfield³⁴ stated that the wall of an abscess begins to form in the first week but it is not firm enough to offer appreciable resistance to an exploratory needle until two to three weeks have passed. The course without drainage depends on the nature and virulence of the organisms. If an insufficient wall or capsule is formed, spreading encephalitis with edema of the brain may quickly terminate the patient's life.

Extension of Infection by Contiguity.—Extension of infection by contiguity is responsible for a large number of brain abscesses. The otologist often encounters an extradural abscess associated with disease of the temporal bone. Cortical abscesses of the temporal and cerebellar lobes resulting from extending infection of the ear occur but rarely. However, accompanying subcortical abscesses of both lobes are frequent, some of which have produced sufficient reaction to seal the meningeal spaces. When this occurs, it is permissible to drain the abscess at the time of mastoidectomy. If the meningeal spaces are not sealed, the introduction of a cannula from an infected mastoid wound into the brain can give rise to another abscess readily. Furunculosis of the scalp, an infected scalp wound, cellulitis, osteomyelitis, and localized

meningitis are frequent sources of cerebral abscess.³⁰ Abscesses resulting from infections of sinuses are owing to inward extensions through diploic channels, emissary, dural, and cerebral veins.

Relation of Frontal Sinusitis to Abscess of the Frontal Lobe.—Woodward,³⁸ in considering the etiology and pathology of osteomyelitis of the frontal bone, has agreed, with others, that the *Staphylococcus pyogenes aureus* is the organism most frequently responsible for the lesion, and has stated that infection of the frontal sinus occludes the ostium and places the pus under pressure. This diminishes the blood supply which, in turn, results in necrosis of the mucous membranes and thrombosis of the perforating veins. From this point, the infection spreads through the diploe, preceded by thrombosis of the diploic veins wherever death of bone has occurred. Pus and granulation tissue fill the diploe, which results in rapid destruction of the osseous elements. Furstenberg²⁰ has regarded the frontal sinus as eroded diploe, the outer wall corresponding to the external plates and the internal wall corresponding to the internal plates of the frontal sinus, which he believes is the chief reason for extension of the infection of the frontal sinus into the diploic spaces. Because the diploic veins communicate with emissary veins, it is apparent how the infection can reach the scalp or dura. If the infection is overwhelming and cannot be stopped by protective reaction, it reaches the cerebral veins with a resulting cerebral inoculation. Extradural abscesses over the frontal lobes are infrequent but occur often enough to warrant consideration when a diagnosis is being made. Abscesses of the frontal lobe do develop from infections of the frontal sinus without accompanying osteomyelitis, just as they develop from infection of the antrum or ethmoid and from infection of the maxilla following extraction of teeth. These infections undoubtedly follow perivascular spaces and thrombosed veins into the subcortical portions of the brain.

Symptoms.—The symptoms of cerebral abscess vary with the different stages of the disease and vary according to the systemic reaction, the degree of increased intracranial pressure, and the situation of the abscess.

Patients who have a temporosphenoidal or cerebellar abscess invariably give a history of acute or chronic otitis media with a suppurating mastoid. Abscesses in the frontal lobe are preceded by acute colds, frontal sinusitis, pansinusitis, and osteomyelitis of the frontal bone. The sudden increase of septic symptoms following a cerebral injury suggests the possibility of a developing brain abscess. Patients become apathetic and an increase in temperature occurs; the temperature remains high for the first few days but soon takes on the septic, steeple-like contour. The number of leukocytes increases to 20,000 or more. Examination of spinal fluid discloses an increase of pressure, increased concentration of protein, and an increase in the number of lymphocytes.³⁷ If active, suppurative meningitis develops, polymorphonuclear leukocytes appear in large numbers, causing the fluid to become cloudy. Also, the active organisms usually are identified.

Headaches and vomiting appear early. Irritability, alternating with stupor

and with rigidity of the neck muscles, is a sign of increased intracranial pressure and meningeal irritation. The pulse is full and bounding; the rate is slower than normal. Choked disks and retinal hemorrhages likewise appear when the normal flow of cerebrospinal fluid has been disturbed.²⁹

The localizing signs depend on the size and situation of the abscess. A temporosphenoidal abscess can produce homonymous defects in the visual fields, homolateral palsy of the sixth and third cranial nerves, and contralateral anesthesia, paralysis, and pathologic reflexes. An abscess in the frontal lobe may attain considerable size without producing localizing symptoms.²⁶ However, I have observed that the initial swelling about one eye is a good diagnostic sign of the lobe affected because the abscess is usually on the same side. Osteomyelitis of the frontal bone invariably extends to one side of the median line, suggesting the lobe involved. Although paralysis may not be present, contralateral reflexes may be exaggerated. When in doubt, it is justifiable to carry out ventricular studies and ventriculography. Cerebellar abscesses produce cerebellar symptoms, plus nystagmus¹¹ and reduction of homolateral reflexes. Hiccough and rigidity of the neck further indicate a lesion of the posterior fossa. Those miscellaneous abscesses that involve the brain stem and pons present bilateral pyramidal signs and bilateral palsy in the distribution of cranial nerves. Those developing from infected wounds are situated in the vicinity of the infection and those developing from nasal sinuses other than the frontal sinus are situated in the lower half of the frontal lobes.

I have seen temporosphenoidal abscesses and one cerebellar abscess produce homolateral pyramidal symptoms with partial contralateral pyramidal symptoms. The symptoms all disappeared following drainage of the abscess. The homolateral pyramidal symptoms were undoubtedly owing to partial displacement of the hemisphere by the abscess, to the opposite side, to such an extent that the crus cerebri was pressed upon and notched by the opposite margin of the tentorium at the incisura tentorii.

The pathologic process, during the initial stage of three weeks, represents a battle between destructive and reparative forces. The secondary quiescent stage, from the second to the fifth week, represents a partial victory for the forces of repair, because the abscess no longer enlarges and the pus is becoming more securely confined by a fibrous capsule. The edema disappears and circulatory disturbance, peripheral to the capsule, is repaired slowly. The recovery will continue up to five weeks, in the case of large abscesses, when apparently it comes to a standstill. During this quiescent stage the symptoms of sepsis subside and the temperature and number of leukocytes recede to normal or slightly above normal. Headaches improve, mental reactions are faster, and the number of cells in the cerebrospinal fluid returns to normal. Localizing symptoms likewise subside but, in spite of the general improvement, periodic headaches occur and choked disks and defects in the field of vision fail to disappear. Reflexes remain exaggerated and motor impairment persists. In cases of abscess of the frontal lobe, symptoms of euphoria or

depression linger. The number of leukocytes continues to rise to 12,000 to 14,000. The patient, on certain days, is apathetic, refuses to eat, and prefers to lie in bed. If it were not for the history of infection and the symptoms of the initial stage, often it would be difficult to distinguish between those symptoms caused by abscess and those produced by a cerebral neoplasm.

Symptoms may continue throughout the quiescent stage for months, until the patient is operated upon, because of an erroneous diagnosis of brain tumor, or dies from rupture of the abscess into the ventricular system or subarachnoid spaces. Small abscesses heal spontaneously with disappearance of all symptoms. Symptoms of abscesses that have been drained surgically disappear, unless important tracts and cortical centers have been destroyed by the inflammatory process. Mutilating operations likewise contribute to permanent injury of cerebral tissue. Epilepsy is a sequela to localized encephalitis and abscess, and it may appear in any case in which there has been a lesion in the frontal, temporal, or parietal lobes. The incidence of this condition may be lowered by proper and adequate drainage. Subsequent resection of the scar may offer some amelioration and relief of epileptic seizures.

Surgical Considerations.—It took many years to learn that performance of a hasty, emergency operation was futile and was accompanied by a high mortality.²¹ This high mortality undoubtedly will follow if the surgeon yields to insistence that he do something as soon as a diagnosis of cerebral abscess has been made.²⁴ Every cerebral abscess passes through a stage of encephalitis before encapsulation occurs. It is during this stage that the infection is virulent and is disseminated most easily. Some surgeons argue that unless the necrotic tissue is removed the patient will die. This is true in the occasional case, and cases have been reported in which aspiration of necrotic material was successful; more often than not, however, the infection is disseminated by surgical intervention and the patient dies from fulminating, suppurative meningo-encephalitis. I believe the best procedure to employ during the acute stage is supportive treatment, rest in bed, high caloric diet, spinal drainage, ice bags to the head, frequent catharsis, moderate amounts of fluids, and, if the patient is comatose, occasional intravenous administration of an hypertonic solution of glucose.

Encapsulation takes place in two to four weeks. The process is an indication that immunity is being established. It is characterized by a decrease in the number of leukocytes to 12,000 to 14,000. The temperature, likewise, returns approximately to normal, 100° F. (37.8° C.), or lower. The number of cells in the cerebrospinal fluid, if increased, returns to normal. The cerebral symptoms gradually subside, but seldom disappear completely until the abscess is drained. Choking of the optic disks, if present, may continue until optic atrophy results. When encapsulation and immunity have been established, thorough and continuous drainage is necessary to effect a cure without recurrence of the abscess.

Surgical Technic.—The ideal exposure of a cerebral abscess is one that allows the surgeon to enter the cranium through a clean field over the ab-

scess.^{2, 3, 6, 8} Exceptions to this rule are when it is desirable to avoid a scar in the frontal region or when it is necessary to pass through a zone of osteomyelitis to reach the abscess. In entering the skull through a clean field, the site is chosen where the abscess is nearest the cortex or which will give the best drainage.¹³ A small incision, 5 cm. in length, is made in the scalp. A craniotomy 3 cm. in diameter is usually large enough to afford ample exposure. The meninges and cortex are then sutured with interrupted stitches of catgut about the margins of the decompression to prevent separation of the cortex from the dura when intracranial pressure is relieved by draining the abscess. The meninges and cortex are further sealed and glued together by use of the electrocoagulating current. A crucial incision is made in the dura to expose an area of brain about 2 cm. in diameter. The margins of the wound are covered with wet strips of cotton in order to minimize the spread of pus between the dura and skull. The cortex overlying the abscess is frequently edematous and cyanotic in appearance.

A round-tipped brain cannula is used to locate the abscess. The resistance of the capsule of the abscess gives one the impression that the cannula is being placed against a flexible, hollow rubber ball. If the abscess has been there for a long time, the resistance may be so great that it is impossible to penetrate the capsule without incising it. Small abscesses may be overlooked, because the firm capsule is capable of deflecting the cannula unless it is directed toward the center of the abscess. As soon as the cannula enters the abscess, the trocar is removed and a Luer syringe, with an intervening rubber connector, is attached to the cannula. The pus is gently aspirated and the cavity is cleansed with small quantities of physiologic saline solution. The cavity of the abscess is explored by incising the cortex and capsule with an electro-surgical needle, using the cannula, which has been left in place, as a guide. The capsule is opened for a distance of 2 cm. in order to insert an illuminated retractor. This makes intracapsular exploration possible, permits further cleansing of the cavity of the abscess, and assures against overlooking pockets communicating with the abscess. During this procedure the capsule is retracted outward against the cortex and skull until the cavity is packed, thus preventing retraction of cerebral structures away from the skull.¹⁸

To assure against collapse of the cortex following drainage of an abscess, I fill the cavity, about the two inserted tubes, with loosely packed strips of iodoform gauze. This continues to keep the capsule moderately distended, instead of allowing it to crumple and give rise to loculated pockets within the capsule. The strips of gauze are shortened daily until they are removed on the tenth day. The gradual collapse and contraction of the capsule prevent recurrence of abscesses and the development of cerebral fungi.

The two tubes are sections of catheters. They are left undisturbed until after the gauze has been removed. One is shortened on alternating days until removed on the twenty-first day; the other is shortened as the sinus closes in and forces it out, which requires from four to six weeks. At operation, the tubes are fastened to the skin to prevent accidental removal during the daily

withdrawal of gauze. Following removal of stitches, the tubes are prevented from falling out by transfixing the exposed ends with safety pins and fastening these to the skin with strips of sterilized adhesive tape. Strips of vaselined gauze are placed over the margins of the wound to prevent the gauze dressings from adhering to the wound. The second tubular drain occasionally is exchanged for a smaller one after the third week but at no time is the cavity irrigated, for fear of disseminating the infection. The patient is allowed to get out of bed after the second day and to leave the hospital after two weeks. His subsequent dressings are done at the office.

Other Technics.—Aspiration by needling has a limited field of usefulness.¹⁷ It is most useful in draining small, sterile, deeply seated abscesses. One aspiration, or two, may be sufficient to drain a sterile abscess but, when the organism still remains active, refilling continues until adequate, continuous drainage has been instituted. There is also danger of spreading the infection by repeated aspiration, as it is impossible to insert and to withdraw the needle through the same tract.

Treatment of the Capsule.—Again I find surgeons divided in their opinions as to the best treatment of the capsule. It must be accepted that if the capsule is left in place, it will result in a fibrous scar. Although this does occur, can it be avoided, and should the capsule be removed at the time of the initial operation or at a later date? Macewen,³¹ Bagley,⁹ Hassin,²³ Cone¹⁴ and many others have demonstrated the pathologic changes that develop to form a capsule about the abscess. From clinical experience, it is apparent that the capsule continues to thicken for several months if the abscess is unrecognized. Therefore, it is fair to assume that the walls will collapse more readily and will be thinner if the abscess is drained properly as soon as immunity and encapsulation have taken place. To assure collapse of the capsule, King^{26, 27} and Cahill¹² have suggested removal of the overlying cortex and the peripheral dome of the capsule. King and others have suggested removal of the capsule at the initial operation. This procedure hastens recovery when it is possible to remove the abscess and capsule without opening it. Bagley reported such a case and I removed such an abscess but was unaware that I had done so until the mass was opened later. The wound, in this case, healed per primam without any drainage. My experience with removal of capsules following evacuation of the pus, however, has not been satisfactory, because secondary suppurative encephalitis develops that is more troublesome to treat than the original infection. If drainage is not instituted, pus becomes inspissated and forms the caseous center of a fibrous mass. The mass will contract gradually until there remains but a small nodule of what once was an abscess, 3 to 5 cm. in diameter. The larger abscesses are more likely to rupture into the sub-arachnoid spaces and ventricular system than the smaller ones; consequently, it is unsafe to wait for these to disappear spontaneously. I cannot concur with those who believe that the rigid capsules will not collapse, for experience has demonstrated that all have collapsed, if properly drained. The difficulty encountered with recurrent abscesses, attributed to failure of the capsule to collapse, I believe is owing to failure to secure adequate and continuous drain-

age. Epilepsy is a symptom and a sequela that will be encountered in the treatment of suppurative diseases of the brain. Medication and dietary regimens offer some assistance in the management of epilepsy. If capsules or scars are to be removed, I believe it is safer to do so after the acute infections have subsided, as practiced by Penfield.³⁴

Cerebellar Abscess.—Cerebellar abscesses have been the most difficult to treat of all cerebral abscesses, because collapse of the cerebellum following drainage frequently results in contamination of the subarachnoid spaces and gives rise to fatal meningitis. Treatment of this group of abscesses has convinced me that the surgically sealed cerebral wound has a useful place in the treatment of brain abscesses. The usual preparation and delay are employed to make sure that encapsulation has taken place. It may be true that the zone of inflammation and adhesion is situated along the sigmoid sinus or petrous bone, but I have found it most advantageous to explore the cerebellum at the most accessible place, which is over the dorsum of each cerebellar lobe. The technic from this point on is similar to that previously described. Electrocoagulation alone cannot be relied upon to seal the meninges and the cortex but must be combined with the use of numerous interrupted stitches of fine catgut to transfix the meninges and cortex around the margins of the limited cerebellar exploration. Leakage of cerebrospinal fluid must not occur. The brain cannula should be directed in an outward, upward direction to avoid entering the fourth ventricle. Otherwise the standard technic is used.

SUMMARY AND CONCLUSIONS

The surgeon should employ supportive measures, such as high caloric diets; also, when the infection is the result of Staphylococci or Streptococci invasion, occasional administration of vaccine and sulphanilamide are helpful.

Osteomyelitis of the skull should be treated similarly to osteomyelitis of other bones; this treatment consists of thorough sequestrectomy and removal of all dead bone. The wound should be cleansed with tincture of iodine and, if drainage is instituted, the drain should be removed within 48 hours and the scalp closed with sutures of silkworm gut.

Mortality will be lowered in the treatment of cerebral abscess if the surgeon employs some of the same principles that are employed in the treatment of suppurative lesions elsewhere in the body.²¹

In cases of suspected cerebral abscess resulting from infections about the ear, with indefinite localizing symptoms, or with localizing symptoms and signs that are conflicting, I have observed the rule of exploring the temporo-sphenoidal lobe before exploring the cerebellum on the side of the infected ear because of the higher ratio of incidence of temporosphenoidal abscess.

If, on study of the physical and neurologic signs, I fail to localize a suspected abscess, performance of cerebral pneumography is justifiable.

Adequate and continuous drainage should be instituted after encapsulation has taken place.

If capsules are to be removed, it is better to remove them after the acute infection has subsided.

REFERENCES

- ¹ Adson, A. W.: The Surgical Treatment of Gummatous Osteitis of the Skull. *J.A.M.A.*, **74**, 385-387, February, 1920.
- ² Adson, A. W.: The Surgical Treatment of Brain Abscess. *J.A.M.A.*, **75**, 532-536, August 21, 1920.
- ³ Adson, A. W.: Pseudobrain Abscess. *Surg. Clin. North Amer.*, **4**, 503-512, April, 1924.
- ⁴ Adson, A. W.: Surgical Treatment of Osteomyelitis of the Skull. *West. Jour. Surg., Obst. and Gynec.*, **41**, 65-77, February, 1933.
- ⁵ Adson, A. W.: Brain Abscess. *Practice of Pediat.*, in press.
- ⁶ Adson, A. W., and Craig, W. McK.: The Surgical Management of Brain Abscess. *ANNALS OF SURGERY*, **101**, 7-26, January, 1935.
- ⁷ Adson, A. W., and Hempstead, B. E.: Osteomyelitis of the Frontal Bone Resulting from Extension of Suppuration of Frontal Sinus; Surgical Treatment. *Arch. Otolaryngol.*, **25**, 363-372, April, 1937.
- ⁸ Adson, A. W., and Pulford, D. W.: Surgical Removal and Pathological Study of a Massive Squamous Cell Epithelioma Associated with Angioma of the Scalp. *Surg., Gynec. and Obstet.*, **42**, 846-848, June, 1926.
- ⁹ Bagley, Charles, Jr.: Brain Abscess: Clinical and Operative Data. *J.A.M.A.*, **81**, 2161-2166, December 29, 1923.
- ¹⁰ Ballance, C. A.: A Lecture on Abscess of the Brain. *Clin. Jour.*, **40**, 273-285, 1912.
- ¹¹ Benedict, W. L.: Abscess of the Brain from the Standpoint of the Ophthalmologist. *Tr. Am. Acad. Ophthalmol.*, 62-53, 1929.
- ¹² Cahill, H. P.: Modern Treatment of Brain Abscess. *J.A.M.A.*, **102**, 273-276, January 27, 1934.
- ¹³ Coleman, C. C.: Some Observations on the Drainage of Subcortical Brain Abscess. *Arch. Surg.*, **10**, 212-216, January, 1925.
- ¹⁴ Cone, W. V.: Personal communication to the author.
- ¹⁵ Craig, W. McK.: Multiple Tumors of the Skull Simulating Osteomyelitis. *Arch. Neurol. and Psychiat.*, **26**, 393-396, August, 1931.
- ¹⁶ Craig, W. McK., and Adson, A. W.: Abscess of the Brain. *Surg. Clin. North Amer.*, **17**, 1077-1091, August, 1937.
- ¹⁷ Dandy, W. E.: Treatment of Chronic Abscess of the Brain by Tapping: Preliminary Note. *J.A.M.A.*, **87**, 1477-1478, October 30, 1926.
- ¹⁸ Dowman, C. E.: The Treatment of Brain Abscess by the Induction of Protective Adhesions Between the Brain Cortex and the Dura Before the Establishment of Drainage. *Arch. Surg.*, **6**, 747-754, May, 1923.
- ¹⁹ Eagleton, W. P.: Brain Abscess: Its Surgical Pathology and Operation Technic. New York, Macmillan Company, 1922, pp. 297.
- ²⁰ Furstenberg, A. C.: Osteomyelitis of the Skull: Osteogenetic Processes in the Repair of Cranial Defects. *Ann. Otol., Rhinol. and Laryngol.*, **40**, 996-1012, December, 1931.
- ²¹ Grant, F. C.: The Mortality from Abscess of the Brain. *J.A.M.A.*, **99**, 550-556, August 13, 1932.
- ²² Gray, Henry: Anatomy of the Human Body. Ed. 18, Philadelphia, Lea and Febiger, 720-721, 730, 1910.
- ²³ Hassin, G. B.: Histopathological Studies on Brain Abscess. *Med. Rec.*, **93**, 91-96, January 19, 1918.
- ²⁴ Horsley, Victor: Case of Cerebral Abscess Successfully Treated by Operation. *Brit. Med. Jour.*, **1**, 636-637, March 24, 1888.
- ²⁵ Kerr, H. H.: Brain Abscess with Especial Reference to Abscess of the Frontal Lobe. *Arch. Surg.*, **7**, 297-305, September, 1923.
- ²⁶ King, J. E. J.: The Treatment of Brain Abscess by Unroofing and Temporary Hernia-

- tion of Abscess Cavity with the Avoidance of Usual Drainage Methods. *Surg., Gynec. and Obstet.*, **39**, 554-568, November, 1924.
- ²⁷ King, J. E. J.: Treatment of Brain Abscess Associated with Extracapsular Necrosis and Suppuration. *Arch. Surg.*, **34**, 631-649, April, 1937.
- ²⁸ Lillie, H. I.: Osteomyelitis of the Cranial Bones Secondary to Paranasal Sinus Operations. *Ann. Otol., Rhinol. and Laryngol.*, **34**, 353-360, June, 1925.
- ²⁹ Lillie, W. I.: The Clinical Significance of Choked Disks Produced by Abscess of the Brain. *Surg., Gynec. and Obstet.*, **47**, 405-406, September, 1928.
- ³⁰ Love, J. G.: Continuous Subarachnoid Drainage for Meningitis by Means of a Ureteral Catheter. *J.A.M.A.*, **104**, 1595-1597, May 4, 1935.
- ³¹ Macewen, William: *Pyogenic Infective Diseases of the Brain and Spinal Cord*. New York, Macmillan Company, 1893, pp. 354.
- ³² McKenzie, K. G.: The Treatment of Abscess of the Brain. *Arch. Surg.*, **18**, 1594-1620, April, 1929.
- ³³ Mayo, C. H.: The Preparation of Dry Bony Areas for Skin Grafting. *ANNALS OF SURGERY*, **60**, 371-372, September, 1914.
- ³⁴ Penfield, Wilder: Epilepsy and Surgical Therapy. *Arch. Neurol. and Psychiat.*, **36**, 449-484, September, 1936.
- ³⁵ Skillern, R. H.: A Case of Extensive Osteomyelitis Involving the Superior Maxillary, Malar, Frontal, Ethmoid and Sphenoid Bone. *Ann. Otol., Rhinol. and Laryngol.*, **29**, 650, September, 1920.
- ³⁶ Williams, H. L.: Osteomyelitis of Frontal Bone Following Incomplete Operation for Acute Frontal Sinusitis. *Proc. Staff Meet. Mayo Clinic*, **3**, 338-340, November 21, 1928.
- ³⁷ Woltman, H. W.: Spinal Fluid Count and Encapsulation of Brain Abscess: an Attempt to Correlate These Factors, and to Determine the Optimal Time for Drainage. *J.A.M.A.*, **100**, 720-722, March 11, 1933.
- ³⁸ Woodward, F. D.: Osteomyelitis of the Skull: Report of Cases Occurring as a Result of Frontal Sinus Infection With *Staphylococcus Pyogenes Aureus*. *J.A.M.A.*, **95**, 927-930, September 27, 1930.

DISCUSSION.—DR. FRANCIS C. GRANT (Philadelphia, Pa.): Doctor Adson has called attention to two very important points in the treatment of osteomyelitis: First, the retention of the pericranium, because from that pericranium, new bone will regenerate. I should like to detail a case which will substantiate Doctor Adson's opinion. I should also like to ask Doctor Adson about his treatment of the more acute osteomyelitic cases. We see, through the nose and throat service, a good many cases of acute frontal sinusitis which develop into acute osteomyelitis of the frontal bone and spread rapidly through the bone. That is the type of case of osteomyelitis which certainly, in our opinion, is very much more difficult to handle than are the chronic cases.

A female, age 12, was admitted to the hospital in 1934, following bilateral frontal sinusitis resulting from diving. She developed an acute osteomyelitis which involved both frontal bones. We had to take off all of her frontal and temporal bones on either side. This was accomplished in two stages, necessarily so, because her condition was poor, but we finally were able to get beyond the edge of the infected bone. These operations were performed in January and February of 1934. After a prolonged convalescence the wounds healed. Skin grafting was necessary.

Roentgenologic studies, in May, 1934, showed that nearly all of both frontal bones and over half of each temporal bone had been removed. But the pericranium had been preserved. Subsequent roentgenograms, in October, 1936, showed practically complete regeneration of the débrided bone. (Case of Gunshot Wound of Head and Case of Osteomyelitis of Skull. *ANNALS OF SURGERY*, **102**, 473-475, September, 1935.)

I should like to ask Doctor Adson one question concerning the treatment of subcortical abscess in the brain. Does he believe in cortical incision with subsequent insertion of packing into the abscess cavity or in simple tap and insertion of tube drainage?

We reviewed 31 cases that were available in the Neurosurgical Clinic, from the standpoint of morbidity, to see which group of patients had the most satisfactory final result.

We believe that the smaller the opening in the cortex to tap, or tap and drain the abscess, the more satisfactory results you will obtain, when these patients are reviewed a year or two later. We found that of 23 cases treated by tap or tap and drain, but five had hemiparesis, convulsions or other symptoms of a serious nature. In eight cases, in which a cortical incision had been made to introduce drainage, seven had a hemiparesis or a history of convulsive attacks.

I am not referring to the immediate mortality but entirely to the morbidity, and my impression is, in the treatment of abscess, if you can handle the case successfully by tap or tapping and tube drainage, the eventual results seem to be very much better.

DR. GILBERT HORRAX (Boston, Mass.): There is just one aspect of Doctor Adson's paper on which I would like to comment, and that is the treatment of the brain abscess. I feel as he does, that we have three available methods, the first of these being the simple one, which Doctor Grant has mentioned, of tapping the abscess and seeing if it is a type with which you can deal in that way. If the abscess is sterile, or in some instances if it is not sterile and can be frequently tapped, this will be sufficient, and I think undoubtedly the sequelae are less.

On the other hand, it has been my experience, as it has that of others, that there are many abscesses in which tapping is not curative. They show signs of increased brain pressure again very soon in spite of frequent tapplings, and one must do something more radical for them.

In our series of 18 chronic abscesses, there were three that were handled successfully by tapping. Many of the others were tapped several times and then we had to do something more serious and more radical with them.

The second method is the open method of drainage which, as Doctor Adson says, was founded by Macewan, and if one goes back to his treatise, as we all do who are dealing with this sort of thing, one will find that his great success was not only the fact that the abscesses were long-standing and well encapsulated, but that he did use this wide open drainage, that is, a relatively wide opening, which Doctor King subsequently adopted in this country in a modified form, and with which he has been so successful.

I think the principle is the same, whether one uses Adson's, King's or Doctor Cushing's method, which consists of marsupialization of the abscess. The latter I have found more successful because many of these abscesses are near enough to the surface and the capsule is of such great strength that you can take sutures through it and sew it to the galea, and thus make a pouch of the cavity and have it entirely extracranial.

I have not seen any cases in which it was necessary to seal off the meninges. I have never seen a case where infection took place by meningitis in that way. It is always by an extension of an osteomyelitic process or by rupture into the ventricle. If the abscess is near enough to the surface, the thing to do, as Doctor King does, is to uncap that area, get down to the abscess and empty a part of its contents. The capsule will then protrude toward the surface, so that one can put in sutures and sew these to the galea, and have a perfectly outside tube, so to speak.

The third method is the one which Clovis Vincent has been advocating so much recently, of extirpating the abscess. We all run into these occasionally and I think that is a method which is very good at times, if the abscess is so situated that one can do it safely; but I do think if one can make use of the simpler methods, the sequelae are going to be less.

DR. JAMES MONROE MASON (Birmingham, Ala.): I was much interested in the remarks concerning osteomyelitis following denudations of large areas of the skull. Some years ago, it fell to my lot to care for two men who, within a few days of each other, received electric burns involving, in one instance, a large area of the scalp over the left parietal bone, and in the other, large areas over the frontal and occipital bones.

The progressive development of the osteomyelitis in the three bones followed along lines which so closely paralleled each other that it suggests what may be expected to take place whenever large areas of the bones of the vault of the skull are denuded of their coverings.

In the burns involving the frontal and occipital bones, the soft parts soon sloughed off and were trimmed away and the underlying bone was dressed antiseptically until such time as the sequestrum should become loosened. This took place within a few weeks, and the loosened bone was lifted off. We found, in each instance, that the entire outer table had become detached, but that the inner table was viable and remained in place except for a small area near the center of the wound. The entire thickness of bones was necrotic and came away with the detached outer table, leaving the dura exposed at this point. The wounds were covered with grafts and the patient recovered.

In the case involving the parietal bone, the same plan of treatment was employed, but the outcome was not so fortunate. About three weeks after the receipt of the injury, the patient suddenly developed hemiplegia and died very shortly from a large brain abscess. Autopsy revealed a similar condition in the progress of the osteomyelitis which we had observed in the other case, namely, that the entire outer table was becoming necrotic, that the greater part of the inner table was resisting the process, but that a point at the center of the involved area of bone was also necrotic. The progress of the infection went entirely through the bone at this point and a small subdural abscess had developed. This quickly extended to the brain and a large abscess was found in the parietal lobe.

Apparently, in large flat bones of the skull the inner table receives sufficient nutrition from vessels surrounding the involved area to insure its vitality when the outer table is denuded, but the circulation near the central part of the inner table may not be sufficiently active for its preservation, and necrosis follows, allowing infection to come directly in contact with the dura.

It is suggested that in large denudations, sections of bone corresponding to the center of the area be removed at once, in order that the tendency to necrosis of the inner table at this point be checked and the danger of the development of brain abscess be lessened.

DR. ALFRED W. ADSON (closing): In reply to Doctor Grant's question, may I state that when the osteomyelitis is limited to the tables of the frontal sinus the lesion is treated by the otolaryngologist. If the lesion extends to the tables of the frontal bone, the operation is divided into two portions. The disease of the frontal sinus is taken care of by the otolaryngologist and the disease of the frontal bone, by the neurosurgeon.

I prefer to seal the meninges to the brain, as described, rather than to employ the technic of extirpation of the abscess, since thus there is less danger of contaminating the subarachnoid spaces. This is especially true of cerebellar abscesses.

RAPID CONTROL OF INTRACRANIAL PRESSURE

ERWIN R. SCHMIDT, M.D.

MADISON, WIS.

FROM THE DEPARTMENT OF SURGERY, UNIVERSITY OF WISCONSIN MEDICAL SCHOOL, MADISON, WIS.

THE present methods for the control of increased intracranial pressure require some time to become effective and depend upon certain physiologic principles. Even if these principles may be questioned on experimental and clinical grounds,^{1, 2, 3, 4, 5, 6} from clinical evidence the mortality rate has declined under this method of treatment. Munro,⁷ in 1925, reported a series of cases collected from the literature with a mortality rate of 37.8 per cent.

Fay⁸ reported a series of cases in which those patients who survived six hours after admission showed a 10.4 per cent mortality, and Ochsner⁹ reports a total mortality rate of 8.4 per cent. By excluding those patients who died in the first 24 hours, a mortality rate of 3.9 per cent was obtained. The method for controlling intracranial pressure we wish to present depends on mechanics alone, is rapid, and we think effective in most cases. Within the cranial cavity there are three substances that are important. Munro¹⁰ pointed this out in 1783, and Kellie,¹¹ in 1824, elaborated the idea. These are the brain substance, the blood vessels with their contained blood, and the cerebrospinal fluid. A change in the volume of one means a change must occur in the volume of one or both of the other substances. The result is that the pressure in the blood vessels, especially the veins, as was pointed out by Grinker,¹² is increased and this pressure causes a rise in the systolic pressure with a slow, full pulse, with an increased pulse pressure, in the systemic circulation. Cushing called attention to the fact that our museums have many examples of fractured skulls, but there were very few examples of injured brains, and that after all the brain itself was more important than the fracture. While our clinical evidence indicates that the mortality rate is improved, little or no attention is paid to the morbidity resulting from cranial injuries.

Lowenberg, Waggoner, and Zbinden,¹³ Caine,¹⁴ Davies,¹⁵ Hahn,¹⁶ and McKean Downs¹⁷ have called attention to the effect of anoxia on the brain in nitrous oxide anesthesia. Lowenberg, Waggoner, and Zbinden report three cases in which there was destruction of the cortex and basal ganglia following the use of nitrous oxide oxygen anesthesia, and one case with clinical evidence of a similar process. These authors point out that death occurs at varying times following the start of the anesthesia, varying from 20 seconds to an hour and a quarter; or it may be hours, days, or weeks before death takes place; or the case may recover with residuals present after years. Signs of anoxemia do not necessarily have to be present. Lowenberg, Waggoner, and Zbinden consider two possibilities: (1) Asphyxia; and (2) toxic effect of the gas. They find support for both ideas, but believe that the toxic effect of the gas is the more likely cause of the destruction.

Courville¹⁸ reports 13 cases, nine of which died. He considers that anoxemia is one of the factors causing changes in the brain and develops a pathology which shows: (a) A sclerosis of scattered pyramidal cells; (b) discrete pale areas in the cortex; (c) patchy necrosis of the superficial, intermediate, deep, or all cortical layers; (d) a subtotal destruction of the cortex; or, (e) if the patient survives for a sufficient interval, a vascular scar may result due to the formation of new blood vessels. Cellular changes are noted in the individual cells. Anoxia has been given little attention. All tissues of the body may be injured, and the degree of anoxia and the length of time necessary to effect damage vary with the individual case. The anesthetist by producing anoxia during the anesthesia can produce symptoms comparable to those resulting from cerebral trauma, and from Caine's case one can see how permanent the damage can be.

The physiologists recognize a critical blood pressure of 80 Mm. of mercury. Clinically, if the systolic pressure remains at this level for 30 minutes or longer, with a very rapid pulse, the patient may be in serious danger. If the pulse is slow, there is no danger. With the development of increased pressure in the cranial vault, the supply of oxygen will vary. The patient may recover, but have irreparable damage of the brain tissue, giving a morbidity which is almost entirely overlooked. The results of the present methods of reducing intracranial pressure require several hours, and extensive damage may take place. There are other times when a quicker relief of intracranial pressure is desirable. This mechanical method was called to our attention, in 1931, by J. R. Learmonth in a case where the increased intracranial pressure was causing vomiting and headache, in a young girl upon whom Learmonth had operated, and found a medulloblastoma. Following the suboccipital decompression and subsequent deep therapy, there was improvement for a time. With the return of pressure, life became unbearable for the patient. A ureteral catheter, No. 6 Fr., was inserted into the ventricle through the old trephine opening, the pressure released, and as long as the drainage was effective, the patient was comfortable.

Fay and Chamberlain¹⁹ have called attention to the fact that if the cerebrospinal fluid can be reduced 1 cc. in volume and kept that way for an hour, it would mean 120 cc. of extra blood to the brain. Ten cubic centimeters' reduction for one hour would mean 1,200 cc. This brings up a number of possibilities when the various problems of increased intracranial pressure are considered, for by means of continuous ventricular drainage, the cerebrospinal fluid volume may be reduced rapidly and for longer periods of time.

The method consists of inserting a No. 6 Fr. ureteral catheter through a trephine opening in the skull. The ventricle is first punctured with an ordinary ventricular puncture needle in order to make a path. Into the ureteral catheter the stylet of the ventricular needle is placed, so as to make it firm. The stylet and catheter are introduced into the ventricle through the previously formed path made by the ventricular puncture. The catheter is anchored with a very fine linen stitch to the skin. The top of the catheter

should be a little above the skin to prevent the skin from closing over the catheter. A 1-2,000 biniodide of mercury dressing is placed over the catheter end. The catheter may become occluded. It is opened by inserting into the catheter a long needle, and then aspirated. After a week or more, suppuration may develop at the skin, and the catheter has to be removed and inserted on the other side. However, the catheter may be kept for longer periods, depending upon the development of suppuration at the skin. We have used this method in 24 cases for the following reasons:

Preoperative reduction of cranial pressure to help stabilize the patient and to prevent sudden pressure changes at the time of operation. Case 14 illustrates this point.

Case Report.—J. W. C. (Hosp. No. 97921), male, age 4, had a posterior fossa lesion. A suboccipital exploration, under novocaine block, was attempted. With the completion of the injection, the patient stopped breathing. When turned on his back, breathing promptly began. Thirteen days later, a general anesthesia was attempted, with the same result. Four days later the patient was placed on his side and a ventricular drain inserted. The cerebrospinal fluid was under great tension. In three days the drainage was less profuse, and ten days later a general anesthesia, intratracheal, was given and a suboccipital exploration revealed a spongioblastoma extending from between the cerebellar hemispheres into the fourth ventricle. He died that night with hyperthermia and signs of medullary failure.

Because of the increased intracranial pressure this patient could not be placed face down. By relieving the pressure, the operation could be postponed, and he could rehabilitate himself. We have used ventricular drainage in patients with high intracranial pressure to give them a period before operation to allow them to habituate themselves, and to obviate the sudden release of intracranial pressure at the time of operation.

Postoperatively we have used ventricular drainage to prevent pressure developing, and making for a smoother convalescence. Fluids are not kept at low levels, and the patient does not need to have repeated injections of hypertonic sucrose or magnesium sulphate enemas.

When operation has failed to remove the cause of the intracranial pressure, ventricular drainage contributes to a smoother convalescence and puts the patient in better condition for radiation therapy.

By relieving the increased intracranial pressure, symptoms and physical signs may develop that will aid in diagnosis, or signs that are due only to increased pressure may disappear.

Horrax suggested that when ventriculography was undertaken, in order to prevent the development of an acute increase in the intracranial pressure, the patient should be operated upon the same day. If symptoms of pressure arise following ventriculography, a catheter inserted into the ventricle will reduce the pressure and there is no need to perform an emergency operation.

Cerebral hernia with separation of wound edges resulting in cerebral fungi may be controlled and the skin edges will heal. Case 2 had to have the bone flap removed because, in spite of all attempts at reducing the pressure, the

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dura could not be closed. The skin was closed, but in several days there was a gaping of the wound with some visible brain substance. Various attempts to get the wound to heal were of no avail. A ventricular drain was then inserted, and in four weeks the skin had completely healed. By a judicious use of ventricular drainage, postoperative hernia may be avoided.

We have not used ventricular drainage in cranial injuries. The other conservative measures have been sufficient, but we would not hesitate to use it when conservative measures failed.

Of the questions that arise as to the complications consequent to employing this procedure, naturally the first is infection. It is a definite risk. Of the 24 cases, six showed definite cloudy fluid. A careful examination of the protocols will show that this is not associated with the length of time the drains were left in. It also shows that a cloudy fluid will clear up (Cases 1 and 15). In the 24 cases, nine are alive and 15 died. Of the six cases with cloudy fluid, their periods of drainage varied from five to 60 days. Two of these cases recovered, one (Case 1) was drained 32 days, and another (Case 15) was drained 39 days. The other cases, 18 in number, were drained from eight hours to 79 days (Table I).

TABLE I
INCIDENCE OF DEVELOPMENT OF INFECTION AFTER DRAINAGE

Case	Infection	Drainage Time	Result
1	Positive—Cloudy fluid 7 days.....	32 days	Recovered
2	Negative.....	40 days	Recovered
3	Negative—(New catheter 8 days P.O.).....	13 days	Recovered
4	Negative.....	10 days	Died
5	Negative (Patient expired same evening. Temperature 106° F. Intracranial pressure not increased).....	8 hours	Died
6	Negative.....	28 days	Recovered
7	Negative.....	8 days	Recovered
8	Positive (16 days after insertion developed parotitis. Culture showed strep., staph., and encapsulated gram-negative bacilli (<i>Klebsiella ozaenae</i>).....	17 days	Died
9	Negative—Catheter lost.....	79 days	Died
10	Negative.....	21 days	Died
11	Negative.....	11 days	Died
12	Positive—22 days.....	60 days	Died
13	Negative.....	9 days	Died
14	Negative.....	10 days	Died
15	Positive—15 days.....	39 days	Recovered
16	Negative.....	12 days	Recovered
17	Positive—10 days.....	18 days	Died
18	Negative.....	14 days	Died
19	Negative.....	3 days	Died
20	Negative.....	6 days	Died
21	Negative.....	10 days	Recovered
22	Positive—6 days.....	7 days	Died
23	Negative.....	25 days	Died
24	Negative.....	22 days	Recovered

There were postmortem examinations in 13 of the 15 deaths. Nine cases showed no evidence of infection; four cases showed definite evidence of infection. Of the four cases with positive fluid findings antemortem, two showed definite evidence of infection; in one case there was no postmortem. Case 12 showed a purulent sphenoiditis with erosion of the sella turcica, brain abscess, and purulent ependymitis; but the pathologists believed this to be secondary to a suppurating sphenoiditis with erosion of the sella turcica. (Patient had an angioma removed at operation.) Case 8 showed nothing. Case 22 showed an empyema of the lateral ventricles, the third ventricle, and a basilar meningitis.

TABLE II
TIME OF DRAINAGE IN POSITIVE CULTURES OF SPINAL FLUID

Case	Development of Infection after Drainage	Result
1.....	32 days	Recovered
8.....	17 days	Died
12.....	60 days	Died
15.....	39 days	Recovered
17.....	18 days	Died
22.....	5 days	Died

Eighteen other cases drained from 8 hours to 79 days. Of 24 cases, 9 recovered and 15 died.

TABLE III
POSTMORTEM EVIDENCE OF INFECTION

Case	Evidence of Infection
4.....	Negative
5.....	Negative
8 (Positive culture).....	Negative
9.....	Negative
10.....	Local meningitis. Fluid clear
11.....	Negative
12 (Positive culture).....	Infection started in purulent sphenoiditis. Purulent ependymitis
13.....	Negative
14.....	Negative
17 (Positive culture).....	No postmortem
18.....	Showed sporotrichosis
19.....	Negative
20.....	Negative
22 (Positive culture).....	Positive
23.....	No postmortem

When we analyze the four cases with positive postmortem findings (Cases 8, 12, 17 and 22), we find Case 8 developed a parotitis that responded to treatment; and, because of high pressure, the drainage was continued. There

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was an exudate over pons, medulla, inferior portion of the cerebellum, and onto the occipital lobes extending forward to the optic chiasma. Case 12 developed secondary to a suppurative sphenoiditis with erosion of the bone, brain abscesses and purulent ependymitis with purulent bronchitis and bronchopneumonia. Case 17 showed scattered granulomatous lesions secondary to a sporotrichosis; and Case 22 showed an empyema of the lateral ventricles, third ventricle, and a basilar meningitis. Case 18, with a parotitis; Case 12, with a suppurating sphenoiditis with extension; and Case 17, secondary to a sporotrichosis, can hardly be attributed to ventricular drainage. Case 22 is directly attributable to ventricular drainage (Tables III and IV). The first

TABLE IV

SUMMARY OF SIX CASES WITH
POSITIVE CULTURES

Two Recovered—Cases 1 and 15
Four Died—Cases 8, 12, 17 and 22

Case 8:

Drained 17 days
Infection developed after drainage 16 days
Parotitis developed 4 days after ventriculography

Case 12:

Drained 60 days
Cloudy fluid after drainage 22 days
Bronchopneumonia after drainage 24 days
Had a purulent sphenoidal sinus with erosion of
bone and extension to cranial cavity

Case 17:

Drained 18 days
Ten days after drainage, cloudy fluid. At same
time developed pulmonary congestion, broncho-
pneumonia and urinary infection
No postmortem

Case 22:

Drained 7 days
Cloudy fluid developed after 6 days
Empyema of lateral and third ventricles
Basilar meningitis

drain ceased to function, and a new drain was inserted through the old pathway. The symptoms of infection and cloudy fluid developed soon afterward. Of the 15 deaths, the 13 postmortems showed no evidence in nine cases. Of the four cases that showed postmortem evidence, only one case can be attributed to the method (Table V).

Repeated roentgenologic examinations of patients with ventricular drains in place have been made, and very little or no air has been found in the ventricles. While attention has been called to the irritating effect of air in the ventricles, we were unable to prove that it played any rôle in ventricular drainage.

Drainage of the ventricles was not possible unless there was increased pres-

sure present. The catheter would become occluded very quickly, which we believe enhances very greatly the entrance of infection into the ventricle.

The infection around the catheter at the skin edge varies a great deal. The length of time the catheter is present has no relation to it. Catheters have been present for many days without any infection developing. In some cases, infection is present as soon as it can develop. We believe the presence or absence of infection depends on dosage, virulence of the bacteria, and the local and general resistance of the patient.

TABLE V
SUMMARY OF POSTMORTEM EVIDENCE
OF INFECTIONS

9 Cases—No postmortem evidence
2 Cases—No postmortem obtained
4 Cases—Positive evidence of infection
4 Cases—Positive cultures
2—Positive postmortem findings
1—No postmortem
1—No findings

In the 24 cases, we lost the catheter in Case 9, due to its slipping into the ventricle, and, at postmortem, it was found to have migrated into the anterior part of the ventricle and imbedded itself for a distance of about 1 cm. into the cerebral tissue. This case was drained 79 days, and the ependyma showed no evidence of inflammation.

Catheters have been fixed by using a fine cambric needle with a linen thread, the suture going through both the catheter and the skin; it is then tied. This has proved to be most satisfactory.

SUMMARY AND CONCLUSIONS

While physiologic methods are available to control intracranial pressure, we present a mechanical method that is not new, for it has been employed in meningitis, to relieve pressure in hydrocephalus, and in reducing pressure preparatory to deep radiation. It is rapid, and in case of the cerebrospinal fluid will reduce the volume as long as the drain works. We have used it in 24 cases and found it to be of aid in reducing increased intracranial pressure preoperatively and postoperatively; to stabilize patients who have had pressure for some time; to aid in diagnosis; to reduce emergency operations after ventriculograms or in acute or high intracranial pressure; and to heal the skin over cerebral hernia. There is a real danger of introducing infection, and, because of this, we feel that this procedure is one that must be used carefully and only after weighing the indications. Cushing's use of ventricular puncture in cases of increased intracranial pressure at the time of operation greatly widened the operative field in neurosurgery. This procedure may aid in its application to a still wider field.

REFERENCES

- ¹ Lehman, E. P., and Parker, W. H.: The Unsolved Problems of Brain Injury: A Critical Review of the Literature. *Internat. Clin.*, **3**, 181, 1935.
- ² Winkelman, N. W., and Eckel, J. L.: Brain Trauma: Histopathology During the Early Stages. *Arch. Neurol. & Psychiat.*, **31**, 956, May, 1934.
- ³ Masserman, J. H.: Intracranial Hydrodynamics: Central Nervous System Shock and Edema Following Rapid Fluid Decompression of the Ventriculosubarachnoid Spaces. *J. Nerv. & Ment. Dis.*, **80**, 138, 1934; Cerebrospinal Hydrodynamics: IV. Clinical Experimental Studies. *Arch. Neurol. & Psychiat.*, **32**, 523, September, 1934.
- ⁴ Browder, J., and Meyers, R.: Observations on Behavior of the Systemic Blood Pressure, Pulse Rate and Spinal Fluid Pressure Following Craniocerebral Injury. *Am. J. Surg.*, **31**, 403, 1936.
- ⁵ Browder, J., and Meyers, R.: Behavior of the Systemic Blood Pressure, Pulse Rate and Spinal Fluid Pressure—Associated with Acute Changes in Intracranial Pressure Artificially Produced. *Arch. Surg.*, **36**, 1, January, 1938.
- ⁶ Pilcher, Cobb: Experimental Cerebral Trauma—The Fluid Content of the Brain after Trauma to the Head. *Arch. Surg.*, **35**, 512, September, 1937.
- ⁷ Munro, D.: Therapeutic Value of Lumbar Puncture in the Treatment of Cranial and Intracranial Injury. *Boston M. and S. J.*, **193**, 1187, 1925.
- ⁸ Fay, T.: Head Injuries: The Results Obtained with Dehydration in 48 Consecutive Cases. *J. Iowa M. Soc.*, **20**, 447, 1930.
- ⁹ Ochsner, Alton, and Hosoi, Kiyoshi: Acute Craniocerebral Injuries. *The Mississippi Doctor*, June, 1935.
- ¹⁰ Munro, Alex: Observations on the Structure and Function of the Nervous System. *Edinburgh*, 1783.
- ¹¹ Kellie: On Death from Cold and Congestion of the Brain. *Edinburgh*, 1824. Reprint from *Trans. Med. Chir. Soc.*
- ¹² Grinker: *Textbook of Neurology*. 2nd ed. Charles C. Thomas, Baltimore, 1937.
- ¹³ Lowenberg, K., Waggoner, R., and Zbinden, Th.: Destruction of the Cerebral Cortex Following Nitrous Oxide-Oxygen Anesthesia. *ANNALS OF SURGERY*, **104**, 801, November, 1936.
- ¹⁴ Caine, A. M.: *Am. Jour. Surg.*, **34**, 103, April, 1920; **37**, 34-36; 89-91, July, 1923.
- ¹⁵ Davies, C. W.: *Brit. Jour. Anaesthesia*, **8**, No. 3, 112, 1931.
- ¹⁶ Hahn, O.: *Zentralbl. f. Chir.*, **58**, 16, 1931.
- ¹⁷ McKean Downs, T.: *ANNALS OF SURGERY*, **99**, 974, 1934.
- ¹⁸ Courville, Cyril B.: Asphyxia as a Consequence of Nitrous Oxide Anesthesia. *Medicine*, **15**, No. 2, May, 1936.
- ¹⁹ Fay and Chamberlain: Personal communications.

DISCUSSION.—DR. ALFRED W. ADSON (Rochester, Minn.): I wish to support what Doctor Schmidt has said. We have had occasion to use continuous ventricular drainage in a number of instances. Occasionally ventricular drainage is performed in connection with a ventriculogram and then it is found that it is impossible to continue with the craniotomy the same day, which I prefer to do whenever possible. In that event, we have introduced a catheter into the ventricle to control intracranial pressure until the next day. This is of special value when there is present an extensive hyperencephalitis owing to a block in the aqueduct or in the region of the fourth ventricle.

At one time we used a ureteral catheter, but during the last few years we have been using a No. 10 French urethral catheter, which is larger and is less likely to become plugged. Thus we diminish the danger of ependymitis by adjusting the catheter. We also have employed it when we have been

called upon to drain a ventricle following craniotomy in cases in which repeated ventricular taps are necessary.

In a number of instances, we have been compelled to resort to continuous ventricular drainage when it was impossible to remove the lesion. On a few occasions we have been able to withdraw the catheter when the block was relieved by irradiation of the tumor.

We have had cases in which ependymitis has developed. I think it has occurred less frequently when the larger catheter has been used. We have also had the catheter slip into the ventricle.

DR. HOWARD LILIENTHAL (New York, N. Y.): I would like to suggest that instead of using a ureteral catheter, which seems to me to be rather a rough sort of thing to use in the brain, a very thin, pure silver catheter be employed, or, if there are objections to that, have a thick-walled cellophane catheter made, which is absolutely nonirritating. It seems to me that these would be preferable to the ureteral catheter, which cannot really be sterilized.

PROGRESSIVE EXOPHTHALMOS ASSOCIATED WITH DISORDERS OF THE THYROID GLAND

HOWARD C. NAFFZIGER, M.D.

SAN FRANCISCO, CALIF.

FROM THE DEPARTMENT OF SURGERY, UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL, SAN FRANCISCO, CALIF.

PRIOR to 1931, a review of the literature showed no concurrence of opinion as to the reasons for the progressive exophthalmos occasionally seen following thyroidectomy. At that time our experience with a severe case resulted in the proposal of an operative treatment which was successful and which permitted an investigation of the pathologic changes involved.^{1, 2} Since then, additional experience has been gained from the observation and treatment of a considerable number of cases, so that now, the pathologic changes involved in this condition have been verified repeatedly by numerous observers, and the value of properly directed surgical treatment is well supported. The interest of many workers relative to the etiologic factors involved has also thrown light on the cause of this serious condition. Our consideration has been given to one particular group, namely, those patients who have progressive exophthalmos associated with disorders of the thyroid gland, and in whom the exophthalmos has progressed to the point at which both the patient's vision and his life are threatened. In the past, in this severely affected—but fortunately small—number, protrusion of the eyes progressed until ophthalmitis resulted, and death invariably occurred from orbital infection and resultant meningitis. Varying degrees and rates of progression of exophthalmos are commonly seen and, in the majority of patients, the proptosis, fortunately, becomes arrested before it reaches an extreme stage.

Many questions remain unanswered concerning the exophthalmos of thyroid disease. For a number of reasons, it has been impossible to secure all the exact information that one might like in order to form an opinion on the relation of exophthalmos to thyroid disease. Clinical records indicate that 50 per cent, more or less, of patients who have exophthalmic goiter present exophthalmos. These figures actually mean little or nothing for the following reasons: A clinical notation of the presence of exophthalmos is ordinarily made when the patient has eyes which appear prominent, with wide lid slits, and a staring expression. By definition, exophthalmos is an abnormal protrusion of the eyeball; but the opinion of even a skilled clinical observer is valueless as to this point except in the most advanced cases, and even then the most experienced are unable to approximate the actual exophthalmos as shown by measurement. In addition, the relation of the eyeball to the orbit shows very wide normal variations. Without knowing the exophthalmometer reading prior to the onset of the disease, one may be unable, even after measurement, to express an opinion as to whether or not there has been abnormal protrusion. Alterations in the position of the eyeball, however, can be fol-

lowed so that progressive changes in its position can be accurately measured and judged. Our own hospital records and the writings of various clinicians expressing opinions as to the presence or absence of exophthalmos in a given series of cases are inaccurate and all but valueless.

Clinical impressions based upon the appearance of the patient are almost entirely dependent upon the width of the lid slits, and changes of several millimeters in the position of the eye with reference to the orbit cannot be appreciated. Even with extreme degrees of exophthalmos, if the lid slits are narrow, a very erroneous impression is gained, and even marked degrees of exophthalmos may not be appreciated at all. Granting that our use of the term exophthalmos is loose and inaccurate, one cannot say much more than that, after an operation for goiter, the apparent exophthalmos disappears in about one-half of the cases and lessens in an additional 15 or 20 per cent. In others it may remain unchanged, but in a few there will be progression of varying grades. This progression may continue slowly, causing no more difficulty than that involved in an unsightly appearance of the patient; progression may halt at any time and it is, fortunately, in only a very small number that it will progress to the danger point.

Previous publications^{1, 2, 3, 4} have referred to and reviewed the varied and ineffective methods of treatment that had been tried, such as sutures of the lids, tarsorrhaphy and canthotomy, various plastic operations on the conjunctiva, incisions of the lids or removal of fat, the Krönlein operation, and section or removal of sympathetic nerves. In connection with the latter, it has been demonstrated that the width of the lid slit is lessened and the appearance of the patient altered by such operations on the sympathetics, but no change occurs in the relation of the globe to the orbit. In a group of patients in whom improvement of the appearance is the aim, a similar result can be achieved by advancing the outer canthus by a suture which will narrow the lid slit. While in animals, stimulation of the sympathetics can produce protrusion of the eyeball through stimulation of the smooth muscle fibers of the orbit, actual protrusion by this means does not occur in man. Sympathetic effects are indicated in human beings only by widening of the lid slits, alteration of the pupil, and vasomotor changes, without alteration of the position of the globe.

From personal communications and reports in the literature, operations for this condition have been performed by Doctors Semmes, Crutchfield, Horrax, Adson, Craig, McKenzie, Teachnor, Oldberg, Ochsner, Dandy, Mixer and White. The cases of these surgeons, with our own eight, make a total of 31. In this series, two deaths were reported and four results were considered poor. One of the poor results was in our own series; operation did not halt the condition, so that later the enucleation of one eye was necessary and the cornea was badly infiltrated in the other. In the patients reported by other surgeons as having poor or unsatisfactory results, it is notable that no eyes were sacrificed and there were no late deaths from meningitis caused by progression of the condition. Considering the hopelessness of these severe

cases of progressive exophthalmos when untreated, the results have been astonishingly gratifying.

In our eight patients who suffered from severe progressive exophthalmos requiring operation, the age varied from 28 to 53 years. Four were females and four, males. All had marked thyrotoxicosis at some time. All had had thyroidectomy for the characteristic symptoms of toxic goiter. These symptoms were relieved by operation but there was no effect on the exophthalmos. Usually, in two months or thereabouts, some progression of the exophthalmos became evident. At the time they presented themselves for treatment, their basal metabolic rates varied from normal to minus 32. Progressive protrusion of the eyes was associated with puffiness of the lids, particularly the upper lids, usually with a large finger-like fold toward the inner canthus. Later, edema of the conjunctiva and limitation of movements of the eyes appeared. About one-half of the patients had varying degrees of choked disk, with hemorrhages. Depending upon the stage of the process, there was a protrusion of mucous membrane from beneath the lower lids. In more advanced stages, corneal ulceration had begun. Vision varied, depending on the condition of the cornea and the stage of involvement of the optic nerve. The highest degree of choked disk was four diopters. In one or two instances altitudinal hemianopsia was present. Before operation, exophthalmometer readings varied from 26 to 34. The lacrimal glands were frequently palpable. Some aching and discomfort referred to the orbit was usually present, increased by efforts to look to the side. All of the patients showed some limitation of movements of the eye muscles, the upward movement of the globes usually showing the most impairment; in only one instance were the downward movements more limited than the upward. Lateral movements were usually greatly affected, frequently reduced about 50 per cent; in others, only faint lateral movement of the globe was possible. Retrobulbar resistance was always greatly increased and readily appreciated. Intra-ocular tension was not altered. Frequently, by raising the lids, the insertions of the recti muscles could be brought into view. In the cases of most marked protrusion, the upper lids could be slipped entirely behind the globe.

Immediately following surgical treatment, there is very marked recession of the eyes. Later, in convalescence, edema of the orbital content appears, causing the eyes again to protrude to about their original position, and there may be an increase in chemosis or protrusion of the mucosa if this was present before operation.

The pathologic changes responsible for progressive exophthalmos of this type seem to be constant. In all of our eight patients, the orbit was tightly packed with the swollen extra-ocular muscles (Fig. 1). These varied in size, being estimated at from five to ten times normal in volume; their color and size depended upon the stage of the pathologic process; often they were pale. The degree of fibrosis varied considerably and the texture of the muscles varied with it. In most cases the muscles were large, slightly paler than normal, firm, and somewhat rubbery. On removing small snips for pathologic

examination, the fibrosis may be readily appreciated and actual hyaline change may be present. In our own eight patients, various minute pieces of tissue were taken from both orbits and from the various muscles, and some 25 fragments were examined. In addition, microscopic examination was made of muscles in 21 patients operated upon by others, and the same pathologic condition was found. In this clinic, Dr. Charles Connor, Professor of Pathology, reported as follows: "The ocular muscles show varying degrees of degenera-

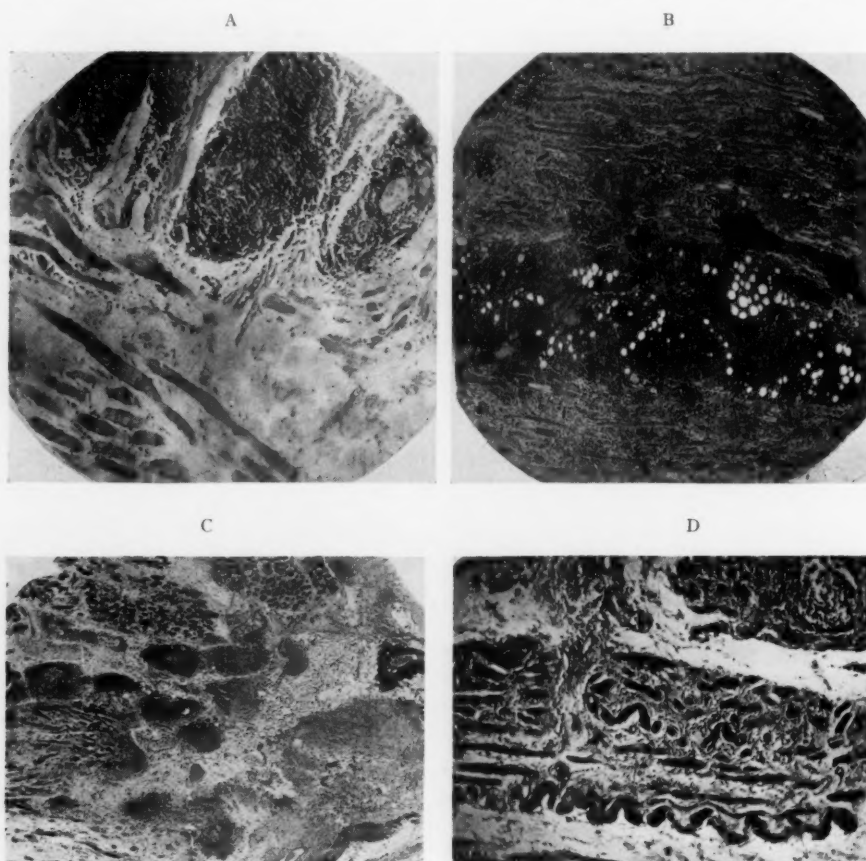


FIG. 1 A, B, C, D.—Photomicrographs showing pathologic changes in the eye muscles in four of our patients.

tion, fibrosis and cellular infiltration. The mildest lesion appears to be a swelling of muscle fibers followed by loss of striation, namely, the early changes associated with so-called Zenker's degeneration. Later, many muscle fibers are seen to be frayed out into a fibrillary substance, which takes the stain for collagen. There is at the same time, in many of the sections, definite interstitial edema. In the more pronounced cases, or perhaps those of longer standing, there is a proliferation of round cells which appears to be coming from the intermuscular mesenchyme. Some of these cells are lymphocytes or immature plasma cells but many are very embryonal in character. Small

and large accumulations are present around the blood vessels. This cellular reaction appears to be, in part, a response to necrosis of the muscle. What appears to be a still later change is the condensation of fibrils into a comparatively dense scar tissue, although in no case can it be said that the condition is resting. One finds early, intermediate and late stages in all of the tissue. In addition to these changes, in some sections the whorls of the arterioles are somewhat thickened and are infiltrated themselves by mononuclear cells. In places, these cells appear beneath the intima causing a slight bulging of the endothelium."

In some of our patients, additional biopsies of muscle were taken from other portions of the body in order to determine whether similar alterations had occurred, but such changes were not found. It is our feeling, however, that they might well be present, particularly in view of such reports as that of Dudgeon and Urquhart.⁵ These writers reported on lymphorrhages in the muscles in exophthalmic goiter in nine cases. The extrinsic muscles of the eye, muscles of the heart, the deltoid, rectus and biceps muscles were examined. One of the patients suffered from myasthenia gravis in addition to exophthalmic goiter. The lymphorrhages varied from a few cells to a wide tract of mononucleated cells, extending for a considerable distance among the fibers and separating them. In some, both small and large lymphocytes were visible. Among the lymphocytes were scattered a few endothelial and plasma cells. Chronic interstitial myositis, with atrophy of the muscle fibers of varying degrees, was a marked feature. In every case, the muscles of the eye were more severely affected than the skeletal muscles and the heart muscle was the least altered. The thymus gland was large. In three cases it showed the usual microscopic appearance met with in this gland in the presence of exophthalmic goiter. In each case, the thyroid gland was characteristic of exophthalmic goiter. The duration of symptoms in these cases was several years. These authors commented that their pathologic findings in the muscles of patients with myasthenia gravis were exactly similar to those described above and that they had not seen the same alterations in other diseases.

In the light of our experiences, it was of particular interest to investigate the literature for other evidences of myopathies. Basedow noted that in one case "because of retrobulbar tension" the recti muscles compressed the globe and caused an indentation between each one. With such an expression, it seems likely that he may have been dealing with enlarged muscles. Numerous writers have commented upon the finding of myositis and great enlargement of the muscles in cases not apparently associated in any way with thyroid disease. In extreme cases in which enucleation was required, large muscles have been commented upon, as they have been in the course of Krönlein's operation. A large number of pathologic reports from cases of idiopathic exophthalmos have been strikingly similar to those in our cases, although, in ours, the association with thyroid disease was clear. Frequently, following an enucleation, the remaining bulk of muscular tissue within the orbit was

sufficiently large to preclude the use of an artificial eye, or required an additional operation to reduce its volume.

The myopathies found in our patients have other special interests. The pathologic changes in the eye muscles are identical with those occurring in the eye muscles of patients with myasthenia gravis. The relation of this disease to disturbances of the thyroid gland and to changes in the thymus have been commented upon by various writers, and the finding of identical alterations in the muscles of our patients may be an additional link in the chain of evidence as to the etiology of these conditions. None of our patients showed recognizable evidence of enlargement of the thymus gland.

Another point which impresses one, in the review of the literature of thyroid disease, is its relation to nervous disorders. In the description of nervous diseases appearing in the course of exophthalmic goiter, one notes the predominance of affections of the cranial nerves controlling the eyes and their movements. More recently articles have appeared under the heading of exophthalmic ophthalmoplegia,^{6, 7} a term applied to a certain group of cases in which there has been no demonstrable disorder of the thyroid gland. There can be little doubt but that the neurologist has frequently sought, in the central nervous system, an explanation for various types of palsies of the eye when myopathy of some of the ocular muscles was responsible. We have observed, and have records of, a large number of patients who had varying degrees of exophthalmos which either was not progressive or did not reach a stage which threatened life and vision, so that operation was not performed. In many of these, the evidence seemed to indicate a myopathy rather than any primary nervous disorder. Some of these gave no evidence of thyrotoxicosis.

While, in most instances, the intensely progressive and dangerous proptosis to which we have applied the term "malignant" had a clear association with a disorder of the thyroid gland and followed operation on this gland, one of our experiences is sufficient to show that this type of exophthalmos may appear under other conditions than following such an operation.

Case Report.—A. D., age 39, referred by Doctor Dowling of Providence, Rhode Island, was admitted to the University of California Hospital in June, 1932, with a history of diplopia in December, 1930, for which glasses were fitted. In March, 1931, his difficulty had increased. In June, 1931, exophthalmos of the left eye was noted. A malignant tumor of the orbit was suspected and roentgenotherapy administered. In September, 1931, however, enucleation became necessary; no malignant growth was found, and the pathologic report was chronic inflammatory disease. Two months later, the remaining eye, the right, began to show exophthalmos. At this time, evidence of thyrotoxicosis appeared. The basal metabolic rate was plus 24, and late in the same month a thyroidectomy was performed. In January, 1932, the basal metabolic rate was plus 6. Nevertheless, the exophthalmos progressed and, by September, 1932, vision was blurred and there was swelling of the optic disk to about four diopters, with retinal hemorrhages.

Upon admission to our hospital, in September, 1932 (Fig. 2A and B), the basal metabolic rate was minus 13. There was great retrobulbar resistance. In addition to the retinal hemorrhages, exudate had appeared. The exophthalmometer reading was 31; the visual acuity was 120/200. Movements of the globe were limited and there was

PROGRESSIVE EXOPHTHALMOS

A

B



FIG. 2 A. B.—Case A. D.: Before operation, October, 1932.



FIG. 3.—Case A. D.: April, 1938. Compare with Figure 2.

contraction of the visual field. Upon elevating the lid, the insertions of the muscles were visible. Operation was performed in October, 1932. There was a reduction of 4 Mm. in the exophthalmometer readings while the patient was still in the hospital. The hemorrhages and swelling of the disk disappeared rapidly and there was improvement in visual acuity. Six years later, the patient considered the eye to be completely restored in vision and movement, although it is still more prominent than normal (Fig. 3).

Such an experience indicates that progressive exophthalmos may precede the development of recognizable thyrotoxicosis as well as appear subsequent to it. In this patient there was a progressive exophthalmos, requiring the



FIG. 4.—Case D. P.: Before operation, January, 1932.

FIG. 5.—Case D. P.: September, 1933.
Compare with Figure 4.

enucleation of one eye, prior to obvious evidences of thyroid disease; in the remaining eye there was progression during a stage of great thyroid activity, but the progressive exophthalmos continued even after other evidences of thyrotoxicosis disappeared.

Pathologic changes similar to those found in progressive exophthalmos must not be assumed for the ordinary degrees of actual or apparent exophthalmos in thyroid disease. There have been but few opportunities for examination of the muscles under such conditions and no uniformity of opinion has been expressed. Such examinations of muscles are needed badly, but

PROGRESSIVE EXOPHTHALMOS

with the low mortality following thyroidectomy, opportunities for postmortem examination have been infrequent.

The operation as previously described by us² does not present any particular technical difficulty and it certainly carries very little operative hazard. In



FIG. 6 A, B, C.—Case S. W.: Before operation, April, 1932.



FIG. 7.—Case S. W.: Immediately following operation.

FIG. 8.—Case S. W.: Ten days after operation.

FIG. 9.—Case S. W.: October, 1933. Compare with Figure 6.

almost all of our patients, the bilateral procedure was undertaken at one stage (Fig. 12), although the wisdom of this is debatable. Following operation, there is immediate recession of the eye. The orbital contents show pulsation. Shortly thereafter, the orbital contents become edematous and the eyes once more protrude. During this period of the convalescence, much care is required. The degree of edema varies greatly. The conjunctivae may have been edematous and protruding from the lids prior to operation. In other

instances, such protrusion may appear after operation and be very slow in subsiding. Any lack of vigilance during this period may result in the drying

A

B



FIG. 10 A, B.—Case C. H.: Before operation, June, 1936.

A

B



FIG. 11 A, B.—Case C. H.: April, 1938. Compare with Figure 10.

of the cornea and result in permanent damage. Great care is required: Scrupulous cleanliness, protection of the eyes with a mild boric, ophthalmic

PROGRESSIVE EXOPHTHALMOS

ointment, and prevention of the drying of the conjunctivae. Following this stage, if the decompression has been adequate, the eyes will be found to have receded from 1 or 2 to 6 Mm., in comparison with the condition before operation. Pulsation of the globe is not appreciated by the patient and tends to decrease and disappear. The extent of the decompression is of the utmost importance, for inadequate decompression is likely only to aggravate the condition. Roentgenologic studies of the frontal and sphenoid sinuses and their relation to the orbital plates and optic foramina are always necessary. The distance to which the frontal sinuses extend into the orbital plate varies greatly, and it is most unfortunate when these sinuses are of unusual size, as the area of bony removal in the anterior fossa is restricted by this anatomic



FIG. 12.—Roentgenogram after operation showing the arrangement of the osteoplastic flaps.



FIG. 14.—Roentgenogram after operation showing optic foramen unroofed.

condition. The same is true, to a lesser degree, in regard to the ethmoids. It is our feeling that, in some of our operations—particularly in the one in which the result was poor—our decompressions were inadequate. For that reason, the type of operation has been altered since our previous publication of the surgical technic.² We now remove not only the area of bone over the roof of the orbit, but carry the removal of bone lateralward into the temporal fossa and up close to the orbital rim, removing that portion of the lateral wall down to the antrum (Fig. 13). Posterior to this, the removal of bone is continued back into the middle fossa, removing the posterolateral portion of the orbit down to, and about, the orbital fissure. When choked disks are present, the optic foramina are unroofed (Fig. 14). This does not entail particular difficulty, and the area of decompression is greatly enlarged. Since we have followed this procedure, the immediate recession of the eyes has been much greater and the postoperative hazard lessened.

As mentioned in previous articles, various experimental workers have re-

ported exophthalmos in animals as being produced by drugs and other substances, particularly endocrine products. Such results need to be scrutinized to determine whether or not sympathetic stimulation alone is responsible for the exophthalmos in animals. In many, at least, that have been studied there is a smooth muscle mechanism which, when stimulated through the sympathetic nervous system, is capable of causing protrusion of the eye. In animals, then, overactivity of the sympathetic nervous system is always a possibility as a cause of exophthalmos, and further studies are needed to determine whether or not other factors are involved. In 1909, Karplus and Kreidl⁸ showed that, in cats, stimulation of the hypothalamus, laterally and slightly posterior to

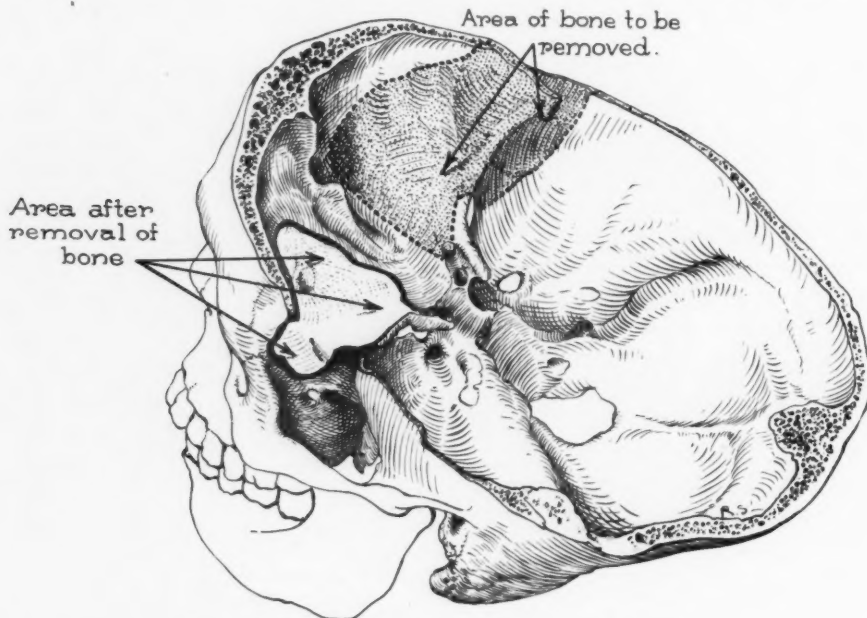


FIG. 13.—Drawing to illustrate the area of bone removed.

the infundibulum, caused dilated pupils, wide lid slits and retraction of the nictitating membrane, proving a true hypothalamic center for the sympathetics. A number of workers^{9, 10, 11, 12} have indicated that the injection of anterior pituitary extract is capable of producing exophthalmos apart from any cause acting through the sympathetic nervous system. Particularly significant work has been done by Smelzer¹³ and Marine and Rosen.^{10, 11} Experimental work has been carried on in our own laboratory* with similar results. We have been able to produce proptosis by the injection of the thyrotropic hormone as produced by the Junkmann process. For this material we are indebted to Dr. Herbert Evans of the University of California. Smelzer recently reported that injections of the thyrotropic, anterior pituitary extract produced exophthalmos in animals from which the thyroid gland and sympathetics had been

* We are indebted to Squibb Company and to Parke, Davis & Company for our supplies of anterior pituitary substance.

removed. He found that the orbital contents became edematous and weighed some 40 per cent more than those of the controls. All of the orbital tissues seemed to participate in this increase in weight, but the extra-ocular muscles in this group weighed some 20 per cent more than those of the controls. He described the tissues as being infiltrated with stainable material, and found the fat to be decreased. Many lymphocytes were present and nests of lymphocytes infiltrated the muscles. He remarked that the microscopic picture is indistinguishable from that in human beings in whom low basal metabolic rates were present. He concluded that the sympathetics have no essential relationship in this process.

Marine and Rosen reported that male rabbits develop exophthalmos more frequently than females. This difference is independent of the thyroid gland, and the exophthalmos develops most frequently in rabbits at about the age of puberty. Gonadectomy greatly reduced the incidence of exophthalmos even in thyroidectomized rabbits. These authors agree that exophthalmos is produced by the thyrotropic hormone and can be caused either by passively introducing this hormone, or by artificially stimulating its production in the animal. Maintenance of normal thyroid activity by the administration of iodine or thyroxin prevents it. They recommended that iodine and desiccated thyroid substance be employed as remedies. It is their hypothesis that a deficiency of some hormone, originating in the suprarenal cortex or gonads, underlies the production of Graves' disease and that the activity of the anterior pituitary is increased by the stimulation of centers in the midbrain.

It is, of course, clear from clinical experience alone that marked and progressive proptosis is not of necessity associated with hyperthyroidism, but more often with hypothyroidism. Sufficient clinical and experimental evidence has been developed to make clear a relationship between these myopathies and other endocrines, and to show that the relationship of the pathologic process to the thyrotropic hormone of the anterior pituitary is intimate.

REFERENCES

- ¹ Naffziger, Howard C.: Progressive Exophthalmos Following Thyroidectomy; Its Pathology and Treatment. *ANNALS OF SURGERY*, **94**, 582, 1931.
- ² Naffziger, Howard C., and Jones, O. W., Jr.: The Surgical Treatment of Progressive Exophthalmos Following Thyroidectomy. *J.A.M.A.*, **99**, 638, August 20, 1932.
- ³ Naffziger, Howard C.: Progressive Exophthalmos after Thyroidectomy. *West. J. Surg. Obs. & Gyn.*, **40**, 530, 1932.
- ⁴ Naffziger, Howard C.: Pathologic Changes in the Orbit in Progressive Exophthalmos. *Arch. Ophthalmol.*, **9**, 1, 1933.
- ⁵ Dudgeon, L. S., and Urquhart, A. L.: Lymphorrhages in the Muscles in Exophthalmic Goiter. *Brain*, **49**, 182, 1926.
- ⁶ Brain, W. R.: Exophthalmic Ophthalmoplegia. *Tr. Ophth. Soc. United Kingdom (Pt. I)*, **57**, 107, 1937.
- ⁷ Stallard, H. B.: A Case of Exophthalmic Ophthalmoplegia. *Tr. Ophth. Soc. United Kingdom (Pt. I)*, **57**, 119, 1937.
- ⁸ Karpus, J. P., and Kreidl, A.: Cited by Marine and Rosen, ref. 10.
- ⁹ Schockaert, J. A.: Enlargement and Hyperplasia of the Thyroids in the Young Duck from the Injection of Anterior Pituitary. *Am. J. Anat.*, **49**, 379, 1932.

- ¹⁰ Marine, D., and Rosen, S. H.: The Exophthalmos of Graves' Disease. Its Experimental Production and Significance. *Am. J. Med. Sci.*, **188**, 565, 1934.
- ¹¹ Marine, D., and Rosen, S. H.: Influence of Gonads on Exophthalmos in Rabbits. *Proc. Soc. Exper. Biol. and Med.*, **35**, 354, 1936.
- ¹² Friedgood, H. B.: Exophthalmos and Hyperthyroidism Produced by Anterior Extracts in Guinea-Pigs; Clinical Course and Pathology. *Bull. Johns Hopkins Hosp.*, **54**, 48, 1934.
- ¹³ Smelzer, G. K.: Experimental Production of Exophthalmos Resembling that Found in Graves' Disease. *Proc. Soc. Exper. Biol. and Med.*, **35**, 128, 1936.

DISCUSSION.—DR. THOMAS M. JOYCE (Portland, Ore.): I have had two of these patients. The first one I saw shortly after Doctor Naffziger's original article appeared; the second one some time later. I would like to ask Doctor Naffziger if he has noticed a pulsation in the eyeballs in his patients, which has persisted and has caused considerable inconvenience to the patient. For some time this patient had difficulty in reading. However, he was able to carry on his work in a garage. The second patient looked very much like the photograph that was shown of Doctor Lahey's patient. My associate, Doctor Kistner, and I noted in the roentgenogram of her skull that she had an unusually large frontal sinus. We took off the inferior plate of the frontal sinus, opened the ethmoid labyrinth and removed the lamina papyracea. The edema disappeared and the patient was absolutely relieved of her trouble.

Note: Since returning home I find that Doctor Kistner has operated upon a second patient which was almost an exact duplicate of this previous case, performing the same operation, namely, removing the floor of the frontal sinus, opening the ethmoid labyrinth and removing the lamina papyracea, with absolute relief.

DR. MARTIN B. TINKER (Ithaca, N. Y.): This condition is distressing because of severe pain to the patient and, as Doctor Naffziger has stated, many of them go on to commit suicide or to death from natural causes. Doctor Naffziger's operation is surely the one indicated in bilateral, severe exophthalmos.

Many years ago, a patient upon whom I had operated for exophthalmic goiter returned with a unilateral exophthalmos. Exophthalmos had not disappeared after thyroidectomy, and the pain had become very distressing. Thinking that there might be intra-orbital pressure, I performed the Krönlein operation, dividing the external angular processes above and below, and the midzygoma, reflecting the wall of the orbit forward to expose the orbital space. A tumor the size of the end of the finger was found pressing upon the optic nerve and the eyeball. It was removed and the result was entirely satisfactory. The exophthalmos disappeared, the pain was relieved and the woman has remained well for many years.

This case was reported at the Section of Exophthalmogy of the American Medical Association. A modified incision was described which was less mutilating than the incision originally proposed by Krönlein. The tumor was examined by Doctor Verhoff, pathologist at the Charitable Eye and Ear in Boston, and was diagnosed as a hemangioma.

It would seem to me that there are perhaps a limited number of cases of unilateral exophthalmos giving very distressing symptoms, in which this operation might be indicated. The bilateral operation, which Doctor Naffziger has described, seems an extremely valuable procedure, and likely to be needed in many cases.

DR. HARRY B. ZIMMERMAN (St. Louis, Minn.): Doctor Naffziger has associated my name with a paper referring to this procedure that was published by Dr. Frank Birch of St. Paul, about the time that this condition first was demonstrated by Doctor Naffziger, probably a little bit before.

The patient, a male, had had a thyroidectomy performed, and shortly afterward developed a unilateral exophthalmos of the right side. When he came under Doctor Birch's observation, the left eye had begun to protrude very slightly. He had a measurable exophthalmos on the left side, but that was not apparent except by actual measurement.

A diagnosis was made of a malignant bulbar tumor, and Doctor Birch and I performed the Krönlein procedure of opening the external plate of the orbit. The tumor immediately presented itself. It was a weird looking tumor, rather diffuse, and consisted of the extra-ocular muscles bulging out into this defect in the external wall of the orbit, a sort of watery, edematous looking muscle. The muscles were incised, and it seemed an interminable procedure to get through the greatly thickened tissue, which was at least almost half an inch thick. That apparently formed the entire tumor, and consisted solely of the enormously thickened, edematous extra-ocular muscles.

The orbit was decompressed by this procedure. The histologic section of the hyperplastic muscles was a good deal as Doctor Naffziger has described. The procedure was stopped. We had some slight regression because of the decompression of the external wall of the orbit, but nothing such as would have been accomplished had the decompression been made as Doctor Naffziger described.

The condition progressed to destruction of that eye, which was eventually removed, by which time he had a loss of the other eye by a similar process. That was the first time it came to my mind that these exophthalmoses were due to this peculiar thickening and hyperplasia of the extra-ocular muscle.

DR. FRANK H. LAHEY (Boston, Mass.): I think it is important to bring out a fact which has not been entirely accepted, and that is that resections of the superior cervical sympathetic ganglia do not produce exophthalmos. Such resection does produce droop of the lid and we have repeatedly utilized it to improve the appearance of some of these patients who have wide palpable fissures. It is, I think, from that point of view, a valuable procedure.

We have had, however, one complaint in connection with that operation and that is that many of these patients who have had resections of their superior cervical ganglia complain of a disagreeable dryness in their nose.

I would like to ask Doctor Naffziger if he has had to utilize suture of the lids, because some of these problems in our hands are pretty acute.

These patients who come in with edema of the conjunctiva are candidates, as you know, for slough. When you realize that the conjunctiva is nourished by osmosis and not by vascularization, it needs only a little increase in lid pressure to produce a slough of the eye covering.

I think, therefore, it is extremely important that you are dealing with intractable exophthalmos to be aware of this danger when you see a wrinkling of the conjunctiva. It has been our experience, and it has been Doctor Horrax' experience as it has been Doctor Naffziger's, that better results have been obtained when more of the bony orbit is unroofed and when the excision is carried down over the outer angles.

DR. HOWARD C. NAFFZIGER (closing): With reference to pulsation of the globes immediately after operation, this is present at once, but gradually lessens and ultimately disappears. The patients are not conscious of it. We

have assumed that a new fibrous covering replaces the peri-orbita and prevents the transmission of dural pulsation.

Diplopia may occur after operation because of the herniation of muscles through the decompression, which temporarily hampers and restricts their movement.

Apparent unilateral exophthalmos associated with thyroid disease is not uncommon. Careful observations have indicated, however, that there is usually some slight degree of exophthalmos in the less prominent eye. In other words, the exophthalmos of thyroid disease is usually bilateral, though there may be marked asymmetry in the degree of protrusion of the two eyes. A purely unilateral exophthalmos is more characteristic of orbital tumor.

The Krönlein operation has been performed for malignant exophthalmos but is inadequate for these severe cases.

Many patients have an unsightly but not dangerously progressive exophthalmos. In these, the widened lid slits need correction more than the proptosis does. This is readily observed by pinching the lids together at the outer canthus, so as to narrow the palpebral fissure; the appearance is greatly improved. Advancing the external canthus by suture is easily performed.

INJURIES TO THE RECURRENT LARYNGEAL NERVE IN THYROID OPERATIONS

THEIR MANAGEMENT AND AVOIDANCE

FRANK H. LAHEY, M.D.,

DEPARTMENT OF SURGERY,

AND

WALTER B. HOOVER, M.D.,

DEPARTMENT OF EAR, NOSE AND THROAT,

THE LAHEY CLINIC,

BOSTON, MASS.

AN INJURY to one recurrent laryngeal nerve during the course of a thyroid operation is an unfortunate incident and an injury to both recurrent nerves is a really serious surgical calamity. Because of the fact that injury to both nerves does not as a rule produce any immediate emergency even though the ultimate results occasion real and crippling disability, also because of the fact that injury to one recurrent nerve causes only temporary loss of voice and usually no limitation in breathing, there has not been as active an interest in the methods for lessening the number of injuries to these nerves as seems desirable.

Thyroid surgery is now so well established upon such a sound basis and with such a low mortality that the operation is being more often accepted by patients and more frequently performed by an increasing number of surgeons. That there is a real need for all of us to interest ourselves in diminishing this incidence of injury to recurrent nerves is established by the fact that even though our percentage ($1\frac{1}{2}$ per cent) of nerve injuries is but one-half of that reported by several other writers (3 per cent), it represents a largely avoidable catastrophe in a great number of patients.

The prevailing attitude of surgeons toward the recurrent laryngeal nerves has been one that has been handed down, unchanged, from the beginning of our experience with thyroid surgery. Kocher, the father of thyroid surgery, advocated partial thyroidectomy under local anesthesia because he could ask the patient to speak and thus know that the recurrent nerves were not injured. This as we now know was not a sound position since in some patients in whom the recurrent nerve has been injured there is not an immediate and striking change in voice. In many patients, immediately after and probably during thyroid operations, there are temporary voice changes without injury to the recurrent nerves. If there is a voice change demonstrating an injury to the nerve, the damage has been done. It is much better to dissect and visualize the nerve and thus avoid its injury. We have all for many years unquestionably accepted what amounts to unsubstantiated impressions about the recurrent nerves, some of which are here recorded: That the recurrent nerves are too small to dissect and demonstrate; that dissection and manipula-

tion, if accomplished, would result in a high percentage of recurrent nerve paralyse; that direct suture of cut recurrent nerves was not worth undertaking because it was thought impossible to find the injured nerves in the scar tissue which forms following subtotal thyroidectomy. It has also been assumed that if the nerves were found, they were too small to be successfully sutured. Because of the above facts it has been considered desirable in thyroid operations not to see recurrent nerves and to avoid seeing them by leaving sufficient strips of thyroid tissue over the nerves so that they would not be visualized.

Some three years ago, one of us (F. H. L.) undertook to demonstrate the recurrent laryngeal nerve in practically all* thyroid operations. This policy has been accepted by all the surgeons in the clinic operating upon patients with thyroid disorders. As a result of this experience, now amounting to well over 3,000 dissections of the nerve at this time, all of the above impressions have been proven to be incorrect. The nerve is of sufficient size so that not only can it easily be dissected but it has sufficient body so that it can be readily palpated as it is pushed laterally against the rigid tracheal wall. It has been demonstrated that routine dissection and demonstration of the nerves and even palpation while on a moderate stretch cause no immediate or late interference with their function. Three nerves previously cut before the patients came to the clinic have been found and accurately and easily sutured. What the eventual outcome will be in these cases done six months and three months ago cannot now be stated. It has been definitely proven, however, that cut nerves can be found and can be sutured without great technical difficulties. As the result of exposing at least 3,000 recurrent laryngeal nerves in a period sufficiently long (three years) to permit late complications to occur if they were to occur, it may be said that the routine exposure of recurrent laryngeal nerves in thyroid surgery is a safe and justifiable procedure and will diminish, if not largely eliminate, injuries to that nerve.†

There have been so few writings and discussions concerning recurrent laryngeal nerve paralyse and the clinical features evidencing this state, that there tends to be considerable confusion about it. One is that if both recurrent laryngeal nerves are injured, there will be immediate difficulty with breathing while the patient is still upon the operating table. Under certain circumstances, later discussed, we believe it is possible for such a respiratory emergency to arise but it is by no means the usual course of events. The clinical history of a patient whose recurrent laryngeal nerves have been interrupted is as follows: During the operation itself, there is usually very little difficulty with the patient's breathing but immediately following operation the patient finds himself unable to talk, due, of course, to the cadaveric position of both cords and the patient's inability to tense them, caused by their

* There will be occasional very ill patients with hyperthyroidism in whom it may prove difficult to find the nerve and in whom the saving of time will be of greater importance than the demonstration of the nerve.

† Up to three years ago the incidence of recurrent laryngeal paralysis was 1.6 per cent. During the last three years, under the above plan, this has dropped to 0.3 per cent.

RECURRENT LARYNGEAL NERVE INJURIES

loss of enervation. Within six months' time, this patient frequently reports to the surgeon quite elated with the fact that her voice is returning and unless the surgeon is familiar with the usual course of events, he too may be elated. In a few months more, however, the patient returns still able to talk but complaining that she is less and less able to breathe satisfactorily, particularly after any unusual activity. This state of affairs progresses still further until, due to the insufficient air-way in the permanently narrowed glottic space,

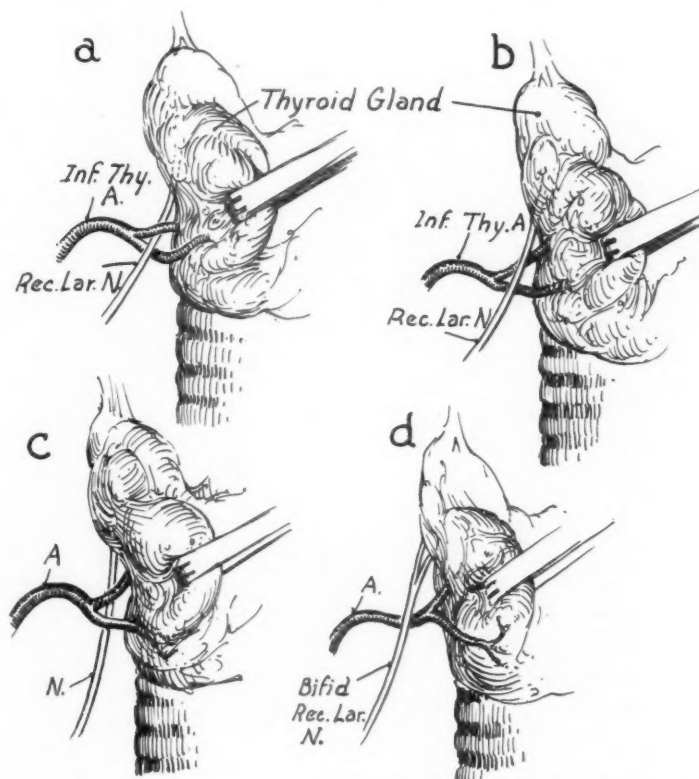


FIG. 1.—Variations in the relationship between the recurrent laryngeal nerve and the inferior thyroid artery encountered in operations on the thyroid.

(a) An uncommon relationship: the nerve passing over one branch of the inferior thyroid and under the other.

(b) Not the rule, but a not uncommon relationship: the nerve passing entirely anterior to the artery.

(c) By far the most common relationship: the nerve passing entirely posterior to the artery.

(d) A not uncommon division of the nerve before entering the larynx.

there is difficulty with breathing on any exertion, even slightly beyond the most moderate.

An additional sign at this time evidences itself also, that is, inspiratory crowing. An excellent way to demonstrate the effect upon breathing of the fixed cords and narrowed glottic space is to ask a patient with bilateral abductor paralysis to count to the highest possible number without taking a breath. It will be found that when the patient has counted from between

15 to 25, a long inspiration will be necessary which, because of the attempt to take in a considerable amount of air, will cause the typical inspiratory crow so characteristic of bilateral abductor paralysis. There is also a characteristic "roaring" during sleep.

A condition under which interference with breathing may conceivably take place during operation would be in a patient most of whose recurrent laryngeal nerves divide extralaryngeally into the abductor and adductor fibers, as shown in Figure 1 *d*, and in whom only the abductor fibers of the nerve were

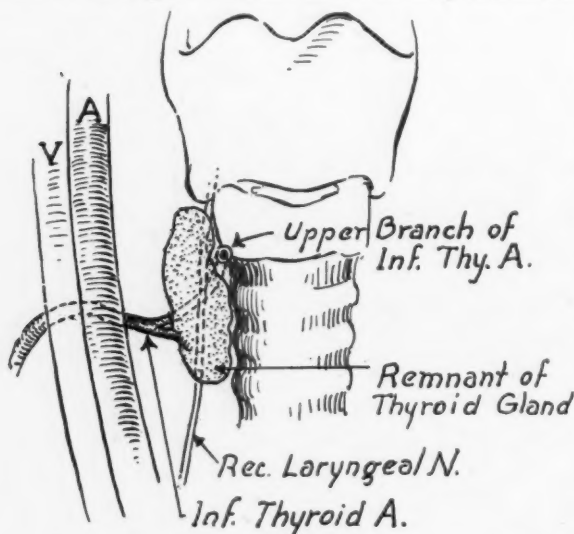


FIG. 2.—Showing the relationship of the recurrent laryngeal nerve, before it enters the larynx, to the upper branch of the inferior thyroid artery. This is depicted diagrammatically as seen from above as it is in a subtotal thyroidectomy. Attempts to snap this bleeding branch in its position between the thyroid and the trachea can result in injury to the recurrent nerve.

The arrow points to the trunk of the inferior thyroid artery, at which point it should be ligated for bleeding of the upper branch of the inferior thyroid artery, rather than attempting ligation of the bleeding vessel itself.

injured at the upper pole. Should this unusual anatomic condition be combined with an unusual bilateral surgical accident in which the injury occurred selectively only in the abductor portion of the bifid nerves, immediate narrowing of the glottic space with difficulty in breathing could then take place.

As the result of an extensive experience with recurrent laryngeal nerves and also based upon the experience which we have had in demonstrating injuries to recurrent laryngeal nerves, we believe that the point at which most recurrent laryngeal nerves are

injured is just below that point where the nerve passes under the lower fibers of the inferior constrictor muscle to become intralaryngeal (Fig. 2).

We have rarely seen injuries to the recurrent laryngeal nerve at the level of the inferior thyroid artery. Anatomically at this level the nerve (Fig. 1), which passes obliquely inward toward the trachea, is usually at some distance from the gland, while at the upper level the nerve is either in contact with that portion of the thyroid gland which rests against the cricothyroid junction or even runs through the thyroid substance at this level (Fig. 2).

We feel very sure that one of the frequent causes of injury to the recurrent laryngeal nerve is the fact that the upper branch of the inferior thyroid artery at this point, as shown in Figure 2, is located between the thyroid gland and either the upper part of the trachea or the cricoid. This vessel is frequently torn off at this point in subtotal thyroidectomies and retracts beside the trachea below the level of the stump of thyroid tissue left behind as the thyroid remnant. One will observe in illustration that if attempts were to be

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made to catch the bleeding end of this vessel, as it retracts between the stump of thyroid tissue and the trachea, the hemostat would frequently be carried well down beside the trachea so that when it catches the bleeding vessel, it may also catch the trunk of the recurrent laryngeal nerve just before it penetrates the constrictor fibers to become intralaryngeal.

We have frequently demonstrated the point of bleeding from this vessel branch by putting a closed hemostat down between the trachea and the remnant of thyroid tissue and then turning the remnant of thyroid tissue inward.

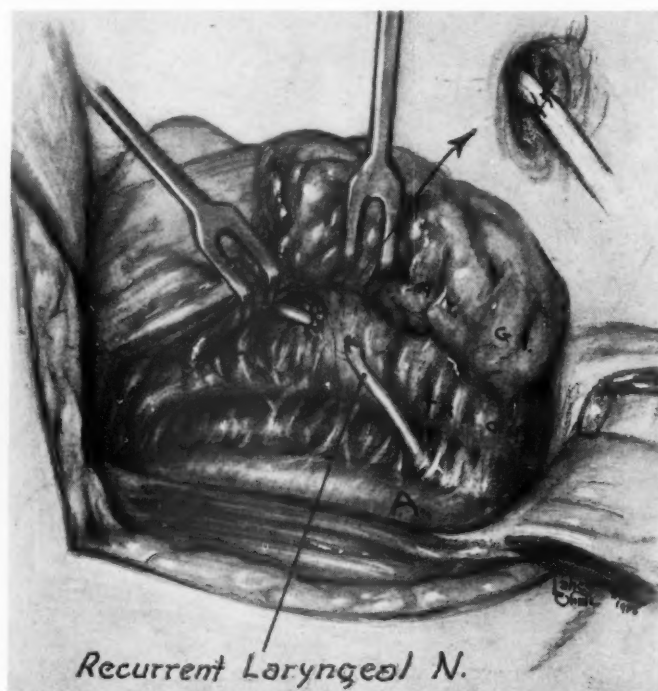


FIG. 3.—This and Figure 4 were drawn at the operating table to demonstrate the location and suture of the recurrent laryngeal nerves in two cases. The nerve had been severed just below the point where it entered the larynx. Note the black silk ties found on each end of the severed nerve.

The insert illustrates the fine black silk vessel sutures inserted on either side of the approximated nerve to oppose fibers conveying similar impulses.

With the recurrent nerve visualized, the close proximity of the nerve to this bleeding point, and the danger of injury to the nerve at this level, become clearly apparent. We feel very sure that technical difficulties with bleeding at this level, which are by no means uncommon, are the cause of many injuries to recurrent laryngeal nerves. Should bleeding occur from the upper branch of the inferior thyroid where it is located close to the trachea at this level, as shown in Figure 2, it is best controlled not by attempting to find the bleeding point but by ligating the trunk of the inferior thyroid artery as it comes up from behind the common carotid at some distance from the nerve, thus protecting the nerve from the danger of injury (Fig. 2). This, we feel

certain, is a valuable and practical point in protecting recurrent laryngeal nerves against possible injury.

Our attitude, and the attitude of surgeons in general, toward injuries to the recurrent laryngeal nerve has, we believe, up to the present been quite wrong. We, as has everyone else, have assumed that if one or both recurrent laryngeal nerves have been injured or cut, nothing can be done about it. Much of this attitude we believe is a traditional one based upon the assumption that severed or injured recurrent laryngeal nerves could not be found and could not be successfully sutured. This we have proven to be erroneous. In an instance (Figs. 3 and 4) in which both recurrent laryngeal nerves had been severed elsewhere ten months previously, the proximal stump on one side having retracted intralaryngeally, both nerves were successfully found and

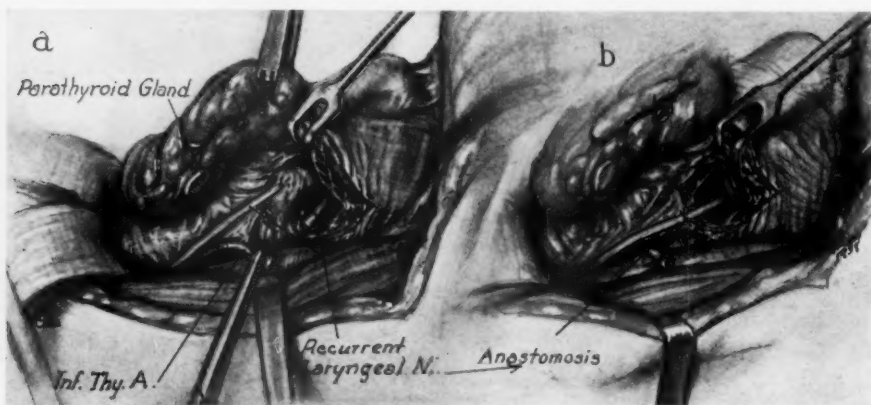


FIG. 4.—(a) The upper end of the severed recurrent nerve had retracted beneath the fibers of the inferior constrictor muscle. Note the black silk tie found on the lower end of the severed nerve. The retracted upper end of the severed nerve was demonstrated by incising the lowest fibers of the inferior constrictor muscle.
(b) Suture of the nerve by two fine silk sutures, attempting to keep abductor and adductor fibers together.

anastomosed without difficulty and without tension with No. 16 China silk on blood vessel needles. In addition to this, in a recent case in which the nerve had also been severed elsewhere, some years previously, in which reoperation in our clinic became necessary because of a recurrent hyperthyroidism, after the removal of the remnant of thyroid, the severed nerve was sought for, both ends were easily found and reunited, merely for the purpose of establishing the technic but with, of course, no prospect of a return of function because of the lateness of the anastomosis.

If we are to successfully reunite severed recurrent laryngeal nerves so that there is a possibility of restoring function to the vocal cords, the operation must be performed, we believe, within three months of the time of injury. It must be appreciated that in late operations, undertaken to resuture injured recurrent laryngeal nerves, not only is there the diminished likelihood of nerve regeneration but also fixation of the arytenoids may well occur. Also, atrophy and fibrosis of the laryngeal muscles may take place, so that even though

impulses pass successfully along the recurrent nerve to the arytenoidius lateralis and the arytenoidius posticus muscles, the fibrotic shortening of the interarytenoid may well prevent the cords from being separated.

As the result of our experience in demonstrating nerves and also because of the ease with which they can be found and approximated, particularly when one makes use of the Berens' binocular loupe, which magnifies the field of vision and the size of the nerve two and one-half times, we believe that in all patients with an injury to a recurrent laryngeal nerve which does not disappear in three months, the nerve should be explored and if injured, the injured section removed, and if severed, the ends refreshed and anastomosed. Delays beyond this time quite definitely jeopardize the probability of a success. Should no injury be demonstrated, exploration will have done no harm, and the patient will have been given the opportunity to recover cord function which might otherwise be completely lost.

When searching for the ends of recurrent laryngeal nerves which have been previously cut, all vascular and scar tissue attachments of the stump of remaining thyroid remnants should first be completely severed from their connection with the internal jugular vein and common carotid artery. The stump of thyroid tissue is then grasped with double hooks, rotated inward and the inferior thyroid artery identified. This is the landmark by which the lower section of the severed nerve may be found as it passes either under or over this vessel as shown in Figure 1 *a*, *b* and *c*. With the aid of the Berens' magnifying loupe, and with a good light brightly illuminating the field, and with care and patience, the stump of the nerve can be found without great difficulty. Since most of the injuries to recurrent nerves, which we have seen, have been close to the point where the nerve enters the larynx, the nerve will usually be found intact at the point where it is in relation to the inferior thyroid artery. The Berens' magnifying lenses, which have a sufficient focal range so that structures can be visualized without danger of the lenses contaminating the field, have for many years proven very useful to us in finding parathyroid bodies, and are especially useful in demonstrating the longitudinal striations of the nerve, thus differentiating it from small veins which can easily be mistaken for it.

The landmark by which the upper section of the severed nerve is found is the horn of the thyroid cartilage. It is just posterior to the point where that structure (thyroid horns) is in contact with the cricoid where the nerve passes beneath the lowest fibers of the inferior constrictors to become intralaryngeal. Unless the nerve has been severed, as happened in one of our cases (Fig. 4), so close to the point where it entered the larynx that it retracts beneath the inferior constrictor, it can usually be demonstrated by finding its point of entrance into the larynx. When the nerve has so retracted, as illustrated in Figure 4, the plan of cutting some of the lowest fibers of the inferior constrictor as suggested by Dr. Charles Frazier, and indicated in Figure 4, will make it possible to find the retracted upper end of the nerve intact beneath the muscle fibers. When found at this point the upper end in

our case possessed enough slack so that it could be pulled down for at least a half inch and was uninvolved in scar tissue.

One of the difficulties of suture of recurrent laryngeal nerves is that of approximating abductor fibers with abductor fibers and adductor fibers with adductor fibers, as it is a nerve carrying impulses to opposing muscles. Since we are as yet able to present only cases in which recurrent nerves have been found and satisfactorily sutured and cannot as yet present proven return of function, we are unable to say whether or not successful restoration of double impulse nerve conduction in a single trunk can ever be successfully accomplished. It may be possible that it will never be effected in humans. One should, however, in anastomosing recurrent nerves, make every effort to unite the nerves so that there is at least a reasonable chance of fibers conveying similar impulses being opposed to each other. When the nerve is visualized through the magnifying lenses it can be seen that it is somewhat flattened, and in uniting it, one should be careful to place the fine silk vessel sutures along the outer and inner margins of the nerve (Fig. 3), one on either side, so that the two ends of the nerve are united in the same flat plane and with no twist in them.

There has been so little experience reported of the surgical approach to this nerve that we can draw only upon our own in discussing what great defects may exist as the result of scarring or excision that require removal of segments of the nerve and still enable one to approximate the cut ends without undue tension upon the nerve and the delicate sutures which unite its severed ends. In the last case in which direct anastomosis was performed, at least a half inch of the nerve was involved in scar tissue and had to be sacrificed. In spite of this loss, the ends could be easily approximated and sutured. The course of the recurrent nerve is obliquely inward toward the trachea. When the nerve passes in front (unusual) of the inferior thyroid artery, there is considerable slack in it. When it passes behind the inferior thyroid artery (its common position), its inward oblique course is somewhat angulated. Severing the inferior thyroid artery between ligatures permits liberation of the nerve from this angulation and provides considerably more length of nerve with which to bridge the defect.

We have, so many times, palpated the nerve, however, when the lobe of the thyroid has been inverted and found it under slight tension, that we doubt very much if it will be possible to make the ends come together when more than one-half to one inch is lost.

If a sufficient section of the nerve has been destroyed so that the procedure physiologically most likely to be successful (direct end-to-end anastomosis) cannot be employed, then, based upon surgical experiences with other nerves, one of two procedures must be resorted to—either the insertion of a nerve graft after the plan proposed by Duell and Ballance in which a section of the distal portion of a nerve which has designedly been cut and permitted to degenerate is inserted, thus serving as a tube along which nerve fibers may

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grow; or the attachment of the end of a foreign nerve to the refreshed proximal end of the recurrent nerve.

This latter procedure was proposed and practiced by Dr. Charles Frazier, who employed the anastomosis of the descending branch of the hypoglossal to the cut recurrent laryngeal nerve, but offers, we believe, little chance of success since the impulses habitually passing down the descending hypoglossal are in no way coordinated with the impulses of respiration. A better chance

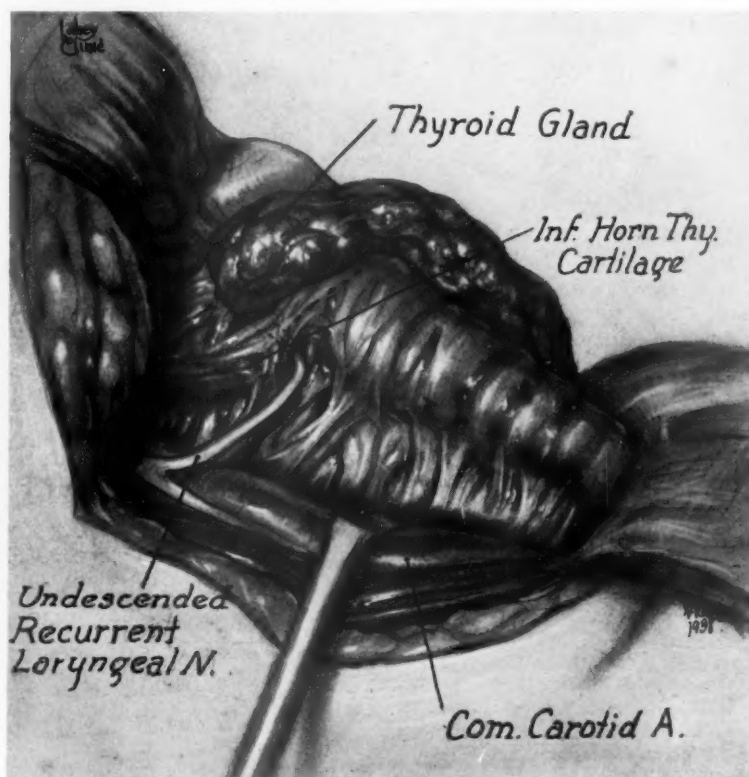


FIG. 5.—Nondescent of the inferior laryngeal nerve. Note how easily such an undescended nerve could be injured if its course were not demonstrated surgically.

for success exists in carrying out the suggestion, made by Mr. Lionel Colledge and Sir Charles Ballance, that a section of the phrenic nerve be anastomosed to the proximal end of the severed recurrent laryngeal nerve, since the respiratory impulses passing down this nerve are coordinated with the impulses constantly passing down the fibers of the recurrent laryngeal nerve. These two investigators, several years ago, performed interesting experiments upon animals relative to the suture of foreign nerves into the cut end of the recurrent laryngeal nerve, and obtained more evidence of a return of function when the phrenic nerve was employed than with the use of any other nerve.

A condition by no means common, but occasionally occurring, is the non-

descent of the recurrent laryngeal nerve. Figure 5 illustrates an instance of such a nondescended inferior laryngeal nerve, which occurred in our own experience. Many anatomists have written on the subject of the nondescent of the inferior laryngeal nerve and a few surgeons, including Dr. John deJ. Pemberton, have described the occurrence of such an abnormality in the recurrent laryngeal nerve.

In the case illustrated by Figure 5, it will be seen that the nerve passes straight across from the vagus to enter directly under the lowest fibers of the inferior constrictor. It will be obvious, in this situation, that if the recurrent laryngeal nerve is not dissected this abnormally located nerve will frequently be caught in ligatures of the upper pole; if, on the other hand, the nerve is sought for as we have advised and is not found, the vagus will be investigated, and this abnormality demonstrated and the nerve thus protected from injury.

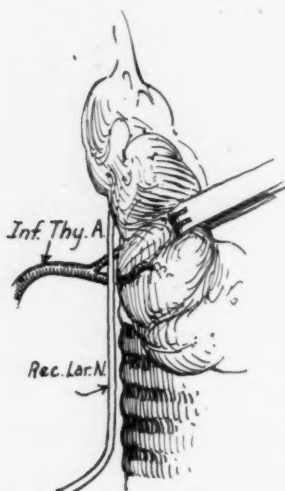


FIG. 6.—The type of displacement of the recurrent nerve toward the trachea which takes place in deep intrathoracic goiters. This is the type of displacement of the nerve commonly found in intrathoracic goiters; and was found to be intact after removal of the intrathoracic goiter shown in Figure 7.

In our early experiences with large and deep intrathoracic goiters, we were quite apprehensive that their removal would result in a high percentage of recurrent laryngeal nerve injuries, on the assumption that there was such an anatomic distortion of the course of the nerve by the large intrathoracic extensions that they would be frequently injured when these deep goiters were roughly extricated from the mediastinum. This has not been the case and repeated dissection and demonstration of the recurrent nerve, after removal of deep intrathoracic goiters, have shown that such distortion of the course of the nerve as does occur, is in a safe direction, displacing the nerve inward against the trachea (Fig. 6), where it is quite safe from injury during the removal of an intrathoracic goiter even of one of such size as is shown in Figure 7.

Thyroid operations have been responsible for 35 instances, or 90 per cent, of bilateral abductor paralysis which have been studied in the Lahey Clinic. In 15 patients, paralysis was temporary and function of one or both vocal cords was recovered. In 20 cases, paralysis was permanent, and of these only one occurred following operation in this clinic. The remainder had been produced elsewhere.

Permanent bilateral abductor paralysis is a real calamity to the individual, because of the marked obstruction of the larynx by the mesial position of the vocal cords, especially on inspiration. We advocate in this condition the employment of a tracheotomy cannula when a patient's activity is limited by want of air. A tracheotomy tube, fitted with a Tucker valve, permits these patients to retain an excellent voice without the inconvenience of having to place the finger over the tracheotomy opening while speaking. This is a

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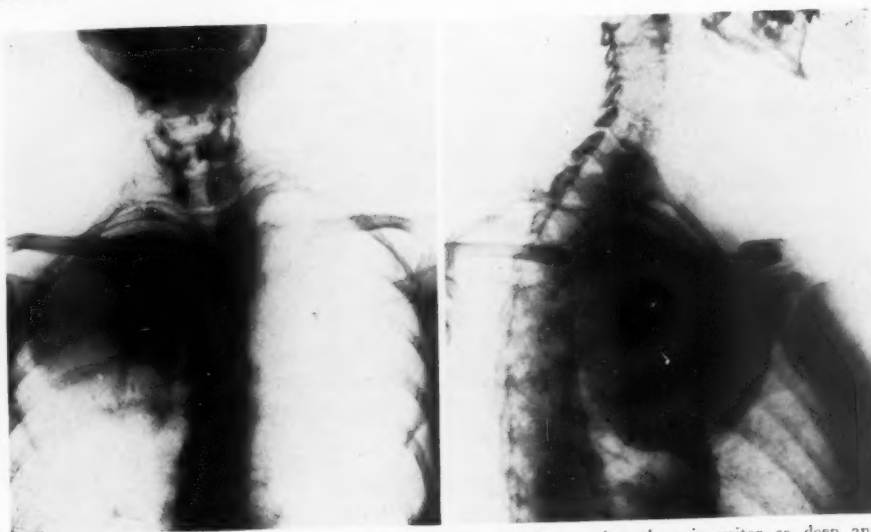


FIG. 7.—Anteroposterior and lateral roentgenogram showing an intrathoracic goiter so deep and so large that its removal could be successfully accomplished only after removal of the inner half of the clavicle and the manubrial portion of the sternum. Dissection of the recurrent laryngeal nerve in this case, as in many other cases of intrathoracic goiter, shows the only distortion of the course of the nerve to be in an inward direction against the trachea (Fig. 6), where it is less likely to be injured by the removal of the intrathoracic extension than when in its normal position.

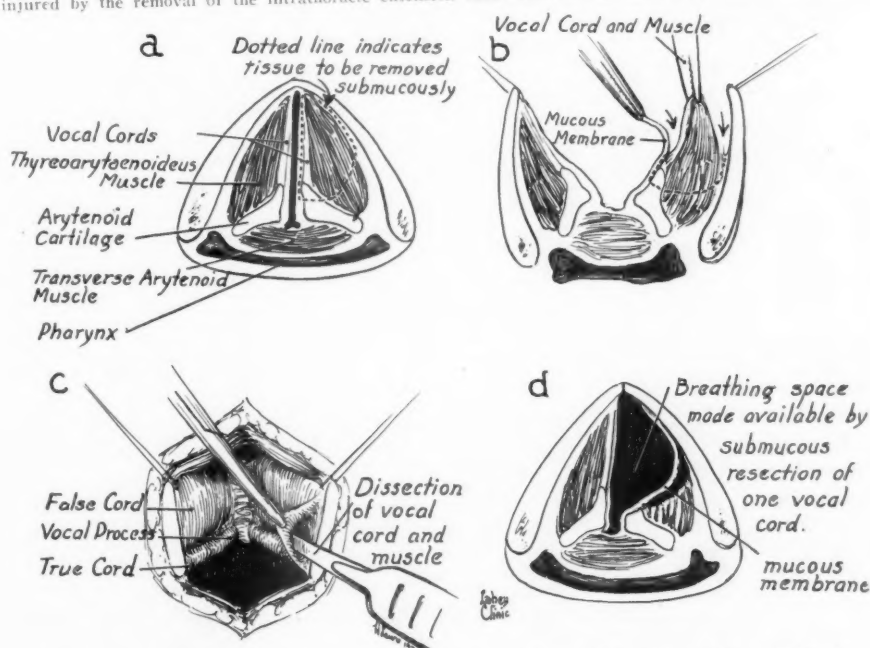


FIG. 8.—Submucous Resection of a Vocal Cord for Bilateral Abductor Paralysis (Hoover).
(a) A diagrammatic cross-section of the larynx in a patient with bilateral abductor paralysis with the median laryngofissure not yet made. Note the area of vocal cord to be resected submucously, demonstrated by the dotted line.
(b) The median laryngofissure made. The mucous membrane in the tip of the hemostat lifted from the cord and muscle.
(c) A front view through the laryngofissure of the dissection of the mucous membrane from over that portion of the cord and muscle to be removed.
(d) A diagrammatic cross-section of the larynx with the laryngofissure closed, showing the cord and muscle removed submucously; the lax mucous membrane pushed back against the laryngeal wall where it is held with gauze packing until it has adhered to the wall and serves as a mucous membrane lining for the enlarged glottic space indicated as breathing space and shown in black.

valuable asset to teachers and saleswomen who depend upon their voice for a living.

A few patients, however, do not become adjusted to the permanent use of a tracheotomy tube and desire to sacrifice their excellent voice for a hoarse voice or whisper if they can but breathe without a tube. In such individuals the submucous resection of a vocal cord is recommended. Eight of the 20 patients with permanent paralysis have been operated upon by this method. The technic of the operation has been previously described by one of us¹ (W. B. H.). A preliminary tracheotomy is performed and followed in five days to one week by median laryngofissure, which gives good exposure of the vocal cords. The mucous membrane is then elevated with a sharp knife over one true vocal cord and above and below the cord as well. The muscle is separated from the cartilage laterally. The muscle is then cut off posteriorly; also the vocal process of the arytenoid is removed. Bleeding is controlled. The mucous membrane is now placed against the lateral wall of the larynx and held in place by an iodoform gauze pack (Fig. 8). The larynx is closed and the pack removed from five days to a week later.

Healing is rapid because there are no raw surfaces to granulate and cicatrize, and the operation leaves an open glottis and an ample air-way. The voice is impaired and is hoarse, comparable to the voice resulting from removal of a vocal cord in cases of carcinoma of the larynx.

In six of the eight cases the results have been quite successful. One patient was infected at the time of operation by vomiting and was not improved. The first patient operated upon was markedly benefited.

As the result of the experience which we have now had with this method of relieving laryngeal obstruction due to bilateral nerve injuries, we feel that the 75 per cent excellent results, obtained up to now, can be still further improved.

CONCLUSIONS

Routine dissection and demonstration of the recurrent laryngeal nerves in thyroid surgery have proven a safe and valuable method of protecting that structure from operative injury, lowering the incidence of nerve injury in three years from 1.6 to 0.3 per cent.

The nerve is of sufficient size to be palpable in many cases.

Dissection and demonstration of the cut ends of accidentally injured or severed recurrent laryngeal nerves, together with accurate suture, is a relatively simple and a very feasible surgical procedure.

Exploration of the recurrent nerves, where injury to these structures has occurred, should be undertaken within three months of the injury if one is to hope for possible restoration of function.

Nondescent of the inferior laryngeal nerve is reported, and such a case illustrated. Other anatomic variations are illustrated.

The advantages of the employment of the phrenic nerve, when foreign nerve anastomoses are considered, are suggested.

A plastic operation on the larynx is described and illustrated which, in six out of the eight cases in which it has been employed, has been successful, by permitting closure of the permanent tracheotomy opening and by providing a natural air-way through the larynx adequate in caliber for any degree of exertion.

REFERENCE

- ¹ Hoover, W. B.: Bilateral Abductor Paralysis; Operative Treatment by Submucous Resection of the Vocal Cords. *Arch. Otolaryngol.*, **15**, 339-355, March, 1932.

DISCUSSION.—DR. WALTER B. HOOVER (Boston, Mass.): Doctor Lahey has said very little about the result of cutting one recurrent laryngeal nerve, and I would like to explain what happens when one recurrent laryngeal nerve is cut, or injured: Usually, there is hoarseness or loss of voice immediately following the section of one recurrent laryngeal nerve, with the loss of function of this one cord. Occasionally, however, the injured cord will lie in the midline of the larynx and there will be no loss of voice, even though one cord is paralyzed. Ordinarily, however, the loss of the function of one cord results in a temporary loss of voice, which lasts from a few days to several months, but one can say almost certainly, that the loss of one recurrent nerve will not result in any discomfort to the patient as far as respiration is concerned, and we can assure the patient that the voice will return, because the cord will eventually come to the midline and will get a very good tension in it with a resulting good voice. This happens no matter what treatment is, or is not, used.

Therefore, we can say that the treatment of the section of one recurrent laryngeal nerve or the loss of function of one cord is that of reassuring or comforting the patient, rather than any other treatment.

Permanent bilateral abductor paralysis is a major catastrophe, because the patient cannot sleep at night, or if he does sleep, is annoying to anyone nearby. The patient is short of breath and cannot exercise, and is truly an invalid, often depressed and despondent. Anyone who has had his activities limited because of bilateral abductor paralysis should have a tracheotomy tube placed. A tracheotomy tube is a safe and certain method of establishing an airway, and every patient, whose respirations are limited or whose activities are limited for want of air must have this done.

The tracheotomy tube should be placed at least as low as the second ring of the trachea so that perichondritis and cicatricial stenosis will not further complicate the picture. A few people are very despondent over the use of a tracheotomy tube, and I know of one instance, in a case where a patient had bilateral abductor paralysis, who refused a tracheotomy tube and died of suffocation. For those patients who are not willing to wear tubes, the operation of submucous resection is reserved. It has been used in only eight cases in our Clinic, six of which have gotten excellent results.

If the patient is a teacher or a saleslady, that is, if she depends on her voice for her living, then the wearing of a tube that has a valve in it, such as devised by Tucker (and there are other tubes on the market), permits them to talk very well; they can breathe well, and their only discomfort is the presence of a tube. Many people are happy with such a tube, but when a person would rather die than wear one, it is justifiable to perform this operation, which does sacrifice the voice in large measure, but not entirely. They at least have a hoarse whisper, very comparable to that of a person who has lost a vocal cord by its removal in carcinoma of the larynx.

DR. FREDERICK A. COLLIER (Ann Arbor, Mich.): Injury to the recurrent laryngeal nerve has always been one of the feared complications of operations on the thyroid and yet it continues to be a very common occurrence—how common one never knows unless there is a routine examination of the larynx before and after the operation.

I have been greatly interested and instructed by Doctor Lahey's discussion of this subject both today and at other times. For years I, too, held to the doctrine I had been taught: that elevation or dislocation of the lobes caused damage to the recurrent nerve, and every effort was made to avoid lifting the lobe until it was freed from the capsule and the thyroid tissue left behind. After an unfortunate experience while performing a total thyroidectomy, I adopted the method I saw practiced by Cutler, of exposing the nerves prior to removing the thyroid. This practice has gradually been extended until we use it frequently but not routinely in subtotal operations.

The recurrent laryngeal nerve, unlike the vagus from which it arises, is a relatively compact nerve with a thin epineural sheath containing little fat. It is in contact with many unyielding structures and tolerates moderate trauma well. It is important to point out the marked differences in the courses of the right and left recurrent laryngeal nerves, as this has a real interest to those operating on the thyroid. The right recurrent nerve splits off the vagus behind the subclavian ~~vein~~ ^{artery} and turns backward and upward around the lower border of the subclavian artery and extends upward along the right border of the esophagus to terminate as the inferior laryngeal nerve. This inferior laryngeal nerve in its course upward is associated with the branches of the inferior thyroid artery sometimes lying between its branches, sometimes in front, sometimes behind them. In its course it extends along the lateral lobe of the thyroid and behind the cricopharyngeal muscle and behind the cricothyroid articulation, divides into a posterior ramus which supplies the cricoarytenoid posterior and the transverse arytenoid muscles—and an anterior ramus supplying the other laryngeal muscles with the exception of the cricothyroid which is supplied by the ramus externus of the superior laryngeal.

The recurrent nerve, on the left side, is given off at the arch of the aorta, it passes around the arch of the aorta and extends upward on the anterior surface of the esophagus to end as the inferior laryngeal nerve.

It can be seen that the differing courses of the right and left nerves offer dissimilar opportunities for injury. The nerve on the right side is shorter and since it lies closely in contact with the right lobe of the thyroid, can be easily injured during operative attacks on the gland, while the left nerve is comparatively protected by its position on the esophagus and is uncommonly injured.

A finding of paralysis of the left cord previous to operation would make one suspicious that the injury to the nerve occurred by a lesion in the thorax such as by aneurysm, mitral stenosis with enlarged heart, pulmonary infection or mediastinal tumor, while this finding on the right would presumptively be due to lesions high in the thorax or cervical region, perhaps a goiter. In our experience, preoperative injury to the nerve due to goiter is rare and when it occurs, not unusually is associated with carcinoma of the gland.

We hurriedly analyzed records of patients seen by Doctor Furstenberg in our Department of Otolaryngology over a period of several months and found 70 patients with paralysis of the cord or cords. Of these, 25 followed operations on the thyroid (in self-defense, may I say none came from our surgical clinic), two were associated with large goiters and two with carcinoma of the thyroid. Of the 25 injured nerves, 18 were on the right side, two on the

left and five were bilateral, demonstrating, I think, the relative chance of injury to the right and left nerves during operation.

We have come to feel that elevation of the lobe, dissections around the lobe, formation of scar tissue following operation are relatively unimportant and infrequent causes of injury to the nerve. The nerve is usually injured by direct attack with hemostat, ligature or knife. We feel that an anatomic exposure of the gland and its surrounding structures tends to prevent nerve injury. This exposure should be especially imperative on the right side where injury is easy and more frequent. On the left side the operator really must go out of his way to injure the nerve.

We have tried very few nerve anastomoses without good results.

Doctor Furstenberg has used a modification of Hoover's operation in which the mucous membrane of the cords is held against the larynx after operation by the superimposition of a two-way, double arm tracheotomy tube. This has, in his hands, produced admirable results following operation for bilateral recurrent nerve palsy.

DR. EMIL GOETSCH (Brooklyn, N. Y.): I would like to say a word regarding injury to the recurrent laryngeal nerve in the treatment of the superior thyroid pole during thyroidectomy, as referred to by Doctor Lahey: We all know that such injuries do take place. It is a common practice, in delivery of the upper pole, to place the finger under the pole and then to elevate it against the tension of the superior thyroid artery. Considerable pressure is, therefore, required and it is during this maneuver that tension is exerted upon the recurrent nerve which lies very near and just medial and behind the pole. As you are aware, the superior thyroid artery divides into a posterior and an anterior branch. It is the anterior branch which is resistant and renders it difficult to elevate the pole. Accordingly, when the latter is large and thick, it is held down in the deep recesses of the neck. Accordingly, to avoid any possible tension upon the recurrent nerve, it has been my practice to free the pole in the following manner: An incision is made into the anterior leaflet of the suspensory ligament at the isthmus, which is then liberated from the trachea and divided. From this point, the avascular space between the larynx and upper pole is entered and the suspensory ligament is clamped, incised and followed upwards until one reaches the superior pole. Thereupon, the anterior branch of the superior thyroid artery as the latter divides at the pole is clamped and divided. This has released the tension holding down the pole, which can then be readily elevated without the necessity of placing the finger behind it. The pole is then resected together with the lobe, and a stump of thyroid gland is allowed to remain anterior to the posterior branch of the artery, which is accordingly spared. During such a procedure there is no tension whatever placed upon the tissues in the region of the pole, and the nerve as it enters the larynx at this level is not traumatized or stretched. By leaving a small stump of tissue adjacent to the posterior branch of the artery, there is a minimum of injury to the smaller vessels in this region and the consequent hemorrhage is practically nil. Deep clamping is therefore never necessary and the occasional injury to the nerve by clamps is also avoided. This method of procedure has been very helpful in the treatment of the upper pole.

I may say a word further on the possibility of injury to the recurrent nerve during or after the resection of large adenomatous goiters which have caused practically complete atrophy of an entire thyroid lobe, leaving a mere film of atrophic capsule between the goiter and the nerve. In the resection or enucleation of such goiters, I have found it advantageous to begin the

dissection of the lobe on the tracheal side, incising the capsule adjoining the trachea and by means of the finger between the goiter and the posterior capsule, rolling the goiter laterally instead of in the opposite direction. The capsule is thus readily recognized and spared and the possibility of injury to the nerve is, I believe, far less likely than in approaching the resection from the lateral side. Whatever tissue is present between the goiter and the nerve is thus kept intact and the occasional instance of hoarseness, four or five months after resection, is avoided.

If the resection is conducted from the lateral side, trauma in the region of the nerve is more likely, and I have felt that the nerve may occasionally be involved in a subsequent fibrosis with consequent functional injury. It is difficult to understand the reason for the late hoarseness on other grounds. Unfortunately, such hoarseness is apt to be relatively permanent as I have seen it in rare instances.

DR. MONT R. REID (Cincinnati, Ohio): In my experience, I have been quite convinced that operating upon goiters with the head and the neck relaxed rather than hyperextended is definitely a safety procedure. By so doing, the gland can be lifted more readily out of the neck, while the nerve tends to drop backward out of the way. When one operates with the neck hyperextended, the muscles, the gland and nerve are taut, which makes it much harder to deliver the gland out of the neck and also increases the chance of injuring the taut nerve while working in a hole.

DR. ELLIOTT C. CUTLER (Boston, Mass.): I would like to add a small anatomic experience to this excellent exposition by Doctor Lahey. During the experience of performing total thyroidectomy on more than 80 patients, we had to expose the recurrent nerves in every case in order to avoid injury to them. When I first attempted the operation of total thyroidectomy, I stood upon the right side of my patient, as had long been my custom in carrying out the operation of subtotal thyroidectomy. In the first 10 cases I twice injured the left recurrent nerve. From that time on I changed my position so as always to operate upon the thyroid lobe from the same side as the lobe, crossing from right to left when I had completed the right side and was ready to attack the left side. The result of this was that no further damage to the recurrent laryngeal nerves occurred.

Would Doctor Lahey, in closing, tell us whether he thinks it is desirable for surgeons performing subtotal thyroidectomies always to cross over when one side is completed, so that the operator will always be on the same side of the patient as is the lobe which is to be removed?

In this large experience with total thyroidectomies I have never found a recurrent nerve lying within the substance of the thyroid gland. This is a finding, of course, in agreement with the embryologic development of the gland and the nerve. They start as separate structures and must necessarily remain separate. I feel certain that the articles which have been published and which make the statement that the recurrent nerve passes into the thyroid gland through its posterior capsule are entirely erroneous and must be based on improper visualization of the structures. Of course, adenomata protruding from the posterior capsule may partially surround a recurrent nerve; but even in such cases the nerve never enters the posterior capsule.

Another matter which I have always thought important in thyroidectomy is the shift of position of the recurrent nerve when the gland is drawn forward sharply by traction. Under such circumstances the nerve, which invariably passes through branches of the inferior thyroid artery close to the

posterior capsule, is pulled forward with the gland. When this dislocation of the position occurs, a clamp hastily placed on the posterior capsule may include the nerve. Doctor Collier spoke of how some surgeons thought it unwise to dislocate the thyroid gland too greatly when removing it. This teaching, I think, emanates from the fact that when one does pull the gland forward, the dangers of catching the recurrent nerve in a hemostat are increased unless the surgeon is willing, as Doctor Lahey suggests, to visualize the nerve and separate the little vessel which is holding the nerve up. It is this same type of technical error that so often results in injury to the common duct. In this latter field, if the ampulla is stuck to the common duct and great traction is exerted, then the clamp, supposedly on the cystic duct, may also include a segment of the common duct.

This matter of the position and the visualization of the recurrent nerve is perhaps the most important knowledge for the thyroid surgeon if he is to avoid trouble. I cannot agree with Doctor Lahey that the nerve must be visualized in every instance; but I do agree that any surgeon who is performing operations upon the thyroid gland should make himself perfectly familiar with the usual position of the recurrent nerve by visualizing it, let us say, in somewhere between 50 to 100 cases.

DR. EDWARD D. CHURCHILL (Boston, Mass.): The recurrent nerves are routinely visualized in operations for hyperparathyroidism, and I think it is fair to say that the nerves are exposed to more trauma and over a longer period of time than in any ordinary thyroidectomy. We have noticed no resulting injury to the recurrent nerves early or late, although a legacy of scar tissue must surround the nerve trunks. In one instance, I inadvertently cut one division of a bifid recurrent nerve and performed an immediate suture. For some reason it was never possible to detect any physiologic disturbance or change in function of the corresponding vocal cord.

An instructive situation was encountered in the case of an opera singer with carcinoma of the thyroid. Her larynx was examined a week before she entered the hospital and found to be normal. The morning after her arrival she said her voice had changed. There was a slight huskiness that I should not have detected but she was very much aware of it, being a singer. Laryngeal examination showed complete paralysis of the right cord.

I suppose that in a great majority of instances, paralysis of a recurrent nerve in association with carcinoma of the thyroid indicates malignant invasion. In this case, we proceeded with an emergency operation on the day that paralysis was first noted. The nerve was isolated and was not found invaded by malignant tissue, but definitely involved in the edematous reaction about a highly malignant tumor.

For her own mental comfort, her larynx was not examined until three months after this operation, when it was found that the right vocal cord moved normally.

The anatomic anomaly of the laryngeal nerve arising directly from the cervical vagus trunk I have encountered once. It is apt to be associated with an anomalous origin of the right common carotid directly from the arch of the aorta and passing transversely across the mediastinum behind the esophagus. This was confirmed by putting a finger down and feeling this anomalous vessel. It can be detected roentgenologically if you know enough to look for it before operation, as a notch in the esophagus is demonstrated by a swallow of barium.

DR. FRANK H. LAHEY (closing): In answer to Doctor Cutler's question, we have never performed a thyroidectomy without crossing to the opposite side of the table. How men can work upon thyroids standing on one side of the table and peering over to look in back in order to see the parathyroids, I have never been able to understand. I do not believe they do. The only way I think you can really see what is in back of the thyroid is to stand on the same side.

I purposely did not mention the question of cutting the prethyroid muscle because it usually causes such an acrimonious discussion. On the other hand, I feel very strongly that if this question of recurrent laryngeal paralysis does exist, and it does, if you can get better exposure by cutting the muscles, and you can, then you ought to cut the muscles. After all, they heal well, and we have cut thousands of them. It does no harm if you cut them high above their innervation.

As to lifting the lobe, I may not understand Doctor Cutler exactly, but I think we have not only lifted the lobe, we have hauled it up out of its bed and turned it upside down, and I do not think it makes one iota of difference. We have taught every surgeon who has been associated with the Clinic to palpate the recurrent nerve by pushing it against the trachea, as it has real body, and can be felt very definitely; even palpation of the nerve under tension does no harm.

Finally, anyone performing thyroid surgery should have a Berens' magnifying loupe. It will magnify two and one-half times, and the focal range is about 18 inches, so that one can keep away from the field and not run the danger of contaminating it. It is a very valuable means by which one can see the striations in nerves and distinguish them from vessels.

AMOUNT OF THYROID TISSUE TO BE LEFT IN OPERATIONS FOR DIFFUSE TOXIC GOITER

MORRIS K. SMITH, M.D.

NEW YORK, N. Y.

IN 1912, William S. Halsted¹ wrote: "Although thousands of operations have been performed, the world over, for the cure of Graves' disease, we are not yet in a position to state how much of the thyroid gland should be removed in any given case." It is still true today. This paper is based on a study of the relation of the estimated size of the gland remnants left at operation to the follow-up results.

It is known from the work of Halsted² and others that removal of a part of the thyroid in experimental animals is followed by compensatory hyperplasia in the remnant. According to DeQuervain³ investigation has shown that at least three-quarters of the gland may be removed without causing hypothyroidism, as long as the gland tissue is healthy. Halsted,¹ in one of his dogs, found that: "Hypertrophy of hardly more than a film of transplanted gland plus, perhaps, the hypertrophy of minute accessory thyroids sufficed . . . to cause the disappearance of myxedema."

In the human subject a very small fragment of normal thyroid may protect against myxedema. Berlin,⁴ in two patients with heart disease, removed approximately nine-tenths of the normal gland. The weight of the normal human gland is estimated at an average of 25 Gm., so that presumably 2 to 3 Gm. remained. There was a drop in metabolic rate and improvement in the cardiac condition, but during the fifth week the metabolic rate began to rise and improvement became less pronounced. Blumgart⁵ and his collaborators, in commenting upon the necessity of total thyroidectomy to relieve damaged hearts, say: "It was not long before it was realized that anything short of complete removal of every vestige of thyroid gland would not permanently lower the basal metabolic rate."

In the early days of thyroid surgery, Kocher and Reverdin discovered the danger of complete extirpation of the gland from the standpoint of subsequent myxedema. It then became a common practice to remove one lobe only, but while many cases of Graves' disease were relieved by this limited procedure, the failures were too numerous and the extensive bilateral resection for toxic goiter gradually evolved with steady improvement in the results.

Thompson⁶ and his collaborators, in their study of postoperative hyperthyroidism at the Massachusetts General Hospital, found that the more experienced thyroid surgeons, who, they observed, did the most complete resections, had a lower incidence of persistent disease than those less practiced. Lahey and Clute⁷ state a commonly held belief: that failure to return to normal is always due to too much hyperplastic tissue in the neck.

The difficulty of defining the proper amount of tissue to leave is illus-

trated by the fact that all surgeons recognize that it varies in different cases, and that many of the most authoritative writers do not go beyond general terms in discussing the matter. Crile⁸ leaves the smallest amount in the severest cases, those with very vascular glands and in originally small glands, but admits that the experience of the surgeon must be the final criterion. Lahey⁹ states that judgment depends on "the degree of involution in the gland, the age of the patient and the degree of improvement with iodine therapy during the period of preparation." He advocates "leaving in fairly good sized remnants in those patients whose glands have involuted well and doing very radical removal in those patients whose glands have not involuted well."

Brenizer¹⁰ leaves a wedge varying from one-quarter to one-half inch in width and thickness. McClure and McGraw¹¹ say that a mere strip of thyroid tissue should be left from the posterior aspect of each lobe. It is noteworthy that they report three times as many instances of postoperative hypothyroidism. Bartlett¹² and Hertzler¹³ advocate a similar procedure in adults. The latter does not fear postoperative myxedema.

Richter¹⁴ is among the most radical as well as the most specific in his description of the amount he leaves. In an article appearing in 1932, he stated that he had been reducing the remnant to less than 2 Gm. but was inclined to leave more, possibly 2 or 3 Gm., because of the persistence of hypothyroidism in too many of his patients. He starts giving desiccated thyroid four weeks postoperative and continues as long as may be necessary, usually several months to a year or more. Most patients return to normal. Out of 447 patients examined after radical thyroidectomy, but one was found to be toxic.

Pemberton¹⁵ represents a more conservative school. He writes: "One of the commonest errors into which many surgeons fall is the belief that recurrence of hyperthyroidism is always directly attributable to inadequate surgical treatment. This had led them to advocate and practice needlessly radical surgery, exacting, as it inevitably must, a higher toll of avoidable complications, such as injury to the laryngeal nerves and production of parathyroid tetany. Today these are far too excessive a price to be paid for the removal of goiter." The operation, as evolved at the Mayo Clinic, includes removal of the isthmus and resection of both lobes, leaving a remnant on each side equivalent to from one-sixth to two-thirds of the amount of tissue in a normal lobe. Expressed in grams this would represent two to eight on each side. As proof that very radical resection cannot guarantee against hyperthyroidism, he cites three cases of postoperative hypothyroidism, two of them definitely myxedematous, in whom exophthalmic goiter eventually recurred.

Ebberts¹⁶ leaves a mass on each side of the trachea equivalent in volume to one-third of a normal lobe, estimated roughly as the size of the terminal phalanx of the little finger. He is less radical where involution is advanced.

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Joll¹⁷ removes no less than seven-eighths of the gland, and in severe cases as much as nine-tenths, and ties all the main arteries.

Estimation of Size of Thyroid Remnant.—For a number of years the writer has been measuring the remnants after they were sutured over at operation by cutting out portions of corresponding size from the specimen removed and weighing them, thus arriving at an estimate of the amount of gland tissue left in the neck. Seventy-five patients with diffuse toxic goiter in whom this procedure has been carried out and who have been followed with metabolism tests for from eight months to six years form the basis of this study. Three of the patients had had a previous resection. Cases of nodular toxic goiter have not been included because it is felt that the diffuse type, in which the tissue changes are of a uniform nature, lend themselves better to a study of this kind.

No one who has attempted to determine the amount of thyroid tissue left at operation is more aware than the writer of the inaccuracies of the method. The weighing has been done after the operation has been concluded, which means that the specimens may have lost in the interval from drying. As a check two observations were made. In the one case a small specimen was weighed in the operating room and again after the operation without notable change. A second specimen which weighed 76 Gm. immediately after removal was found to be 68.5 Gm. on standing until after the operation was completed. This, however, is not a serious source of error, as the proportion of the remnants to the size of the gland is small. More important is the fact that the remnants are irregular in shape, and while they are measured in three planes, one is aware that the blocks cut out from the specimen are at best a rough approximation. Finally, there may be extensions of the remnant which the operator fails to appreciate. Nonetheless, with all the objections, the method offers a more concrete basis for judgment than visual or tactual appraisals alone.

There have been no deaths during the period of this study which would allow of securing at autopsy and weighing the actual remnant as a check on the estimate. The writer has, however, carried out the procedure in the autopsy room. Such observations indicate that small to medium remnants have probably been underestimated by about 2 Gm.

Of the 75 patients studied, ten, or 13 per cent, showed postoperative hyperthyroidism. Another has had a basal metabolic rate that is often somewhat above normal but as she has had no symptoms she is not considered recurrent. Of the ten cases, eight are continuations. Of the two remaining, one was definitely hyperthyroid at the end of a year and the other, two years. Metabolism tests obtained in the interim on the first were +11 and +15 per cent; on the second, +27 per cent, so that these patients may not have been completely relieved of their toxicity at any time.

The estimated size of the remnants has ranged between 3 and 25 Gm. Thirty-seven were recorded as 6 Gm. or less and 38 as 7 Gm. or more. If 2

Gm. are added as a correction, the division would come between 8 and 9 Gm. instead of between 6 and 7—probably closer to the actual amounts.

The proportional size of the remnant was calculated by adding its corrected weight (estimated weight plus 2 Gm.) to that of the specimen, and dividing the result, the weight of the whole gland, by that of the corrected remnant. Forty were found in this way to have one-seventh, or less, of their gland remaining, and 35 one-sixth or more, the proportions ranging from one-twentieth to one-third.

Factors Other Than Size of Remnant Influencing Result of Treatment.—

If all cases in the series were identical the study of the effect of the different sizes of remnant on the outcome of the operation would be much simplified. Of the variables which have to be considered, that of primary importance is the severity of the disease. In order to take account of this factor the series has been divided into two, on the basis of elevation of basal metabolism before treatment. As would be expected, the great majority of patients with postoperative hyperthyroidism came from those in the higher range, eight as opposed to two in the lower range. Size of the gland is a factor of at least technical importance. There were more large glands among the severer cases.

Involution has been mentioned as a factor in judging the amount of gland tissue to leave. In this series the writer has not recognized any notable trend on the basis of iodine response. Those in whom the metabolism fell to below +20 per cent, on their preoperative iodine, had a very low incidence of postoperative hyperthyroidism. They were, however, almost altogether drawn from the milder cases.

Three patients of the series were adolescents, age 13 and 14, too few to furnish any conclusions as to treatment in the young. In general, however, the writer prefers to be conservative in resections in this age-group. The oldest patient was age 60.

It is now proposed to examine the effect of the size of the remnant considered from the two aspects of weight and proportion on: (1) The general metabolic outcome of the series; (2) the incidence of postoperative hyperthyroidism; (3) the incidence of postoperative hypothyroidism; and (4) individual cases.

Size of Remnant and Metabolic Result.—A composite metabolism progress chart has been prepared of the two groups of the series based upon the weight of the remnants. Persistent and recurrent cases are included until such time as they may have been submitted to roentgenotherapy or reoperation (Table I).

It is seen from analyzing Table I that there is no apparent difference in the collective outcome, although the group with lesser remnants contains a larger proportion of milder cases, as is also evidenced by the lower average of first metabolism tests.

A similar chart has been prepared based upon the proportion of the remnants, with the correction of 2 Gm. added, to the size of the gland (Table II).

In the division, as seen in Table II, the group with proportionally smaller

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remnants contains a much larger proportion of the severer cases, which is reflected in the higher average admission metabolism. There is nothing to choose in the collective aftercourse.

TABLE I
COMPOSITE METABOLIC RATE

	Six Gm. or Less		Seven Gm. or More	
	Corrected, 8 Gm. or Less		Corrected, 9 Gm. or More	
	B.M.R.	No. of Cases	B.M.R.	No. of Cases
Admission.....	+51	37	+62	37
Preoperative.....	+23	37	+28	38
Postoperative.....	+ 1	35	+ 2	34
3 months.....	+ 6	21	+ 4	24
6 months.....	\pm 0	26	\pm 0	28
1 year.....	\pm 0	28	\pm 0	33
2 years.....	\pm 0	24	+ 2	22
3 years.....	- 5	15	+ 2	14
4 years.....	+ 5	9	- 2	13
5 years +.....	- 4	9	- 3	10

TABLE II
COMPOSITE METABOLIC RATE

	Remnant			
	One-Seventh or Less		One-Sixth or More	
	B.M.R.	No. of Cases	B.M.R.	No. of Cases
Admission.....	+65	40	+46	34
Preoperative.....	+30	40	+20	35
Postoperative.....	+ 3	37	+ 1	32
3 months.....	+ 5	26	+ 6	19
6 months.....	+ 1	32	- 1	21
1 year.....	- 1	34	+ 1	27
2 years.....	+ 2	24	- 2	20
3 years.....	- 3	18	- 1	10
4 years.....	+ 2	13	- 2	7
5 years +.....	- 4	9	+ 1	9

In order to approach the question from a somewhat different aspect, the series has been divided into three groups: (1) Those whose postoperative basal metabolism tests have averaged less than ± 0 , 35 in number; (2) those who have averaged above ± 0 but have not been considered hyperthyroid postoperatively, 30 in number; and (3) the ten with persistent and recurrent hyperthyroidism.

As between groups (1) and (2), the former contains a larger proportion of mild cases. The average size of remnants is the same. Group (3), made up of the ten persistent and recurrent cases, contains a preponderance of originally more toxic patients, while the size of the remnants averages 10.5 Gm. as opposed to 7 in the remainder of the series.

Size of Remnant and Incidence of Postoperative Hyperthyroidism.—Approaching the subject now from this aspect, one finds that in the half of the series with smaller remnants by weight the incidence of postoperative hyperthyroidism is 8 per cent as opposed to 18 per cent in those with larger rem-

nants. The former group, however, contains 62 per cent of milder cases which modifies the significance of the figures. Dividing the series into thirds, that with smallest remnants has fewest failures.

Where the series is divided into two, on the basis of corrected proportional size of the remnants, there are an equal number of persistent and recurrent cases in each group. The result must be considered better, however, in the group with smaller proportional remnants, as it contains 70 per cent of severer cases. Dividing the series into three, the third with smallest remnants proportionately includes only one patient with persistent hyperthyroidism, although it is made up largely from the severer cases.

Among the ten failures in the series studied, seven had remnants above the median in weight and three below. Of these three below, all were from relatively small glands and fell among those with the largest remnants proportionately. It would seem that a reduction in thyroid tissue that was thorough, both from the point of view of actual as well as proportional size, should be followed by a minimum of postoperative hyperthyroidism.

Size of Remnants and Postoperative Hypothyroidism.—There are six patients who have been given thyroid medication for a period but none have had to remain on it permanently. These six include both severer and milder cases as well as those with smaller and larger remnants. It is apparent, however, that postoperative hypothyroidism may ensue despite a good sized remnant of thyroid tissue.

Size of Remnant in Individual Cases.—When the persistent and recurrent cases are reviewed individually one feels, in almost all instances, that had the resection been more radical the result would have been better.

ILLUSTRATIVE CASES

Case 1.—Mrs. C., age 27, married had a primary basal metabolism rate of +55 per cent. Seventy-nine grams of thyroid tissue were removed. The remnant was estimated to be 12 Gm., considerably above the average of the series. Three months after operation the metabolic rate was +41 per cent, and she was not well. She had roentgenotherapy and responded promptly. Since then her tests have been within normal limits, the last one —6 per cent, nearly six years after operation.

Case 2.—Mrs. P., age 48, was hypertensive as well as hyperthyroid, and presented a different aspect of the problem. Her primary metabolism rate was +71 per cent. The gland was a small one, the specimen weighing 17 Gms. and the remnant being estimated at 6. On the seventh postoperative day the metabolism was +5 per cent, but by the end of four months it had risen as high as +36 per cent. She received roentgenotherapy, which completed the cure as far as we have been able to observe her. In this instance the remnant was small by weight but large in proportion to her original supply of gland tissue.

However, what seems like an adequate resection will not always insure a satisfactory result.

Case 3.—Miss R., age 30, had a primary metabolism rate of +50 per cent, and a second of +41 per cent before treatment. The specimen weighed 28 Gm. and the remnant was estimated at 5. Although the patient gained 20 pounds and looked well, she had a metabolism rate at nine months of +20 per cent and at 11 months of +28 per

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cent, accompanied by symptoms of fatigue and emotional instability. On iodine her metabolism rate has remained within normal limits during the past few months but she still has symptoms.

In this instance the remnant was small, and while its proportion to the size of the gland was greater than the median of the series, the operator felt that he had, if anything, gone too far, as the toxicity was not severe and the gland of normal size.

Case 4.—Mrs. S., age 37, shows that the patient may do well as could be wished despite the fact that she has been left with considerable thyroid tissue. On admission her basal metabolism rate was +64 per cent. At operation the estimated remnant was large by weight, 11 Gm., and also proportionately to the size of the goiter. Postoperative metabolism tests have been at six months, +1 per cent; at one year, -9 per cent; and at four years, -13 per cent.

Case 5.—Mrs. P., the only one of the series to come to reoperation thus far, is presented to illustrate regeneration. At the first operation the specimen weighed 74 Gm. and the remnant was estimated at 9. At the second operation, two years later, 19 Gm. were removed, leaving an amount estimated at 5 Gm.

SUMMARY AND CONCLUSIONS

The power of regeneration of the thyroid gland is very great. The accumulated experience of surgeons has been that resections for toxic goiter must be extensive in order to keep the incidence of unsatisfactory results at a low figure, although many successes have been recorded after what is now considered inadequate surgery.

In the series under review the writer has attempted to perform thorough but not extremely radical resections. The record of 13 per cent relapses, a figure which may well be increased after a longer period of observation, in some of the more recent cases particularly, would argue that, in general, criticism might point rather toward too great conservatism than otherwise.

One cannot place too much confidence on statistics based upon such rough methods of estimation as have necessarily been employed, yet it is felt that they have some value.

The most important factor in planning the extent of operation is the severity of the disease. Size of the gland, both for its bearing on technical difficulties as well as on the relative proportion of remnants to be left, is also important.

Studies on the collective outcome of the series would seem to indicate that, within the limits of resections performed, there was not much to choose between larger and smaller remnants. When, however, the incidence of persistent and recurrent cases is used as a criterion, the results favored the smaller remnants both on the basis of actual weight and relative proportion. Examination of the individual failures showed that in no case did the remnant fall into the smaller half of the series on both counts. This indicates that not only must the operator plan on leaving a small remnant but that he must be guided in his judgment by the size of the original goiter and in the case of small very toxic glands be particularly radical. This is only logical when

one recalls that an equally severe grade of intoxication may be found in the presence of glands of very different sizes.

On the basis of this study, the writer thinks that a remnant of 4 or 5 Gm. as he has estimated it, or as corrected, 6 or 7 Gm., should result in fewer failures. To present the matter more concretely, blocks of tissue measuring 3x1x1 cm. from two specimens of hyperplastic iodized glands were weighed and found to be 2 to 2½ Gm. This amount on each side of the trachea seems a satisfactory amount to allow to remain in the more toxic cases unless the gland is small. In the case of medium to good sized glands, a remnant of 5 Gm. would represent a small proportion of the original goiter, say one-tenth or less, a relative amount which in the series studied was followed by a low incidence of postoperative hyperthyroidism. In the case of glands which are little larger than normal, despite a marked toxicity, one should leave smaller amounts than usual so that the relative reduction of thyroid tissue is thorough. In large glands with only mild toxicity, on the other hand, more generous remnants ought to meet the indications.

REFERENCES

- ¹ Halsted, W. S.: Report of a Dog Maintained in Good Health by a Parathyroid Autograft Approximately One-Fourth of a Millimeter in Diameter, and Comments on the Development of the Operation for Graves' Disease as Influenced by the Results of Experiments on Animals. *J. Exper. Med.*, **15**, 205, 1912.
- ² Halsted, W. S.: An Experimental Study of the Thyroid Gland of Dogs, with Especial Consideration of Hypertrophy of This Gland. *Johns Hopkins Hosp., Rep.*, **1**, 373, 1896.
- ³ DeQuervain, F.: *Goitre*. Translation by J. Snowman. Wm. Wood & Co., New York, 140, 1924.
- ⁴ Berlin, D. D.: Therapeutic Effect of Complete Thyroidectomy on Congestive Heart Failure and Angina Pectoris. *Am. J. Surg.*, **21**, 173, 1933.
- ⁵ Blumgart, H. L., Riseman, J. E. F., Davis, D., and Berlin, D. D.: Therapeutic Effect of Total Ablation of Normal Thyroid on Congestive Heart Failure and Angina Pectoris. *Arch. Int. Med.*, **52**, 165, August, 1933.
- ⁶ Thompson, W. O., Morris, A. E., and Thompson, P. K.: Thyrotoxicosis Following Subtotal Thyroidectomy for Exophthalmic Goiter. *Arch. Int. Med.*, **46**, 946, 1930.
- ⁷ Lahey, F., and Clute, H. M.: Persistent and Recurrent Hyperthyroidism. *ANNALS OF SURGERY*, **83**, 199, 1926.
- ⁸ Crile, G., and Associates: *Diagnosis and Treatment of Diseases of Thyroid Gland*. W. B. Saunders Co., Philadelphia and London, 409, 1932.
- ⁹ Lahey, F. H.: The Technique of Subtotal Thyroidectomy for Hyperthyroidism. *Surg. Clin. North Amer.*, **16**, 1587, December, 1936.
- ¹⁰ Brenizer, A. G.: Amount of Gland to Be Left at Thyroidectomy. *ANNALS OF SURGERY*, **97**, 831, 1933.
- ¹¹ McClure, R. D., and McGraw, A. B.: Postoperative Hypothyroidism. *Western Jour. Surg., Obst. and Gynec.*, **39**, 690, September, 1931.
- ¹² Bartlett, W.: *Surgical Treatment of Goiter*. C. V. Mosby Co., St. Louis, 210, 1926.
- ¹³ Hertzler, A. E.: *Diseases of the Thyroid Gland*. C. V. Mosby Co., St. Louis, 160, 1935.
- ¹⁴ Richter, H. M.: Thyroidectomy for Thyrotoxicosis. *Surg., Gynec. and Obst.*, **54**, 551, 1932.
- ¹⁵ Pemberton, J. de J.: Recurring Exophthalmic Goiter. *J.A.M.A.*, **94**, 1483, May 10, 1930.

DIFFUSE TOXIC GOITER

- ¹⁶ Ebberts, E. M., with the Assistance of Fitzgerald, R. L., and Silver, P. G.: *Surgical Diseases of the Thyroid Gland*. Lea and Febiger, Philadelphia, 130, 1929.
- ¹⁷ Joll, A. C.: *Diseases of the Thyroid Gland*. William Heinemann, London, 518, 1932.

DISCUSSION.—DR. WILLIAM BARCLAY PARSONS (New York): Doctor Smith's interesting paper brings up certain points of technical and philosophic importance that are worthy of comment. In my opinion, it is doubtful if we are entitled to assume that the behavior of the gland remnant left behind after operation on a pathologic gland will be similar to that of normal thyroid left by mistake in attempting total thyroidectomy for heart disease. The former would be far more subject to either regressive or progressive change, while the latter would have a tendency toward normal behavior.

One naturally agrees with all the general principles laid down by Doctor Smith. One cannot help having some doubt as to the estimation of the weight of the remnant, particularly if one has had experience in the technic. In 1930 and 1931, Gutman, at the Presbyterian Hospital in New York, was interested in estimating the total weights of thyroids in the course of some iodine studies he was pursuing. We estimated the gland remnant for him by measuring the remnant before suturing, which should be accompanied by less error than after suturing. To indicate roughly how much a fresh piece of thyroid weighs, one flat pear-shaped piece measuring $1\frac{1}{8}'' \times \frac{7}{8}'' \times \frac{1}{4}''$ weighed 6 Gm.; another measuring $1'' \times \frac{7}{8}'' \times \frac{1}{8}'' - \frac{1}{4}''$ weighed 4.5 Gm. Pieces of this size are by no means large, but were adequate for these two particular cases, as they were in satisfactory condition when last seen, six and seven years after operation. We really had but little confidence that our measurements came within an error of 10 per cent, and it is probable that we all leave far more tissue behind than we believe we do. Fortunately in the vast majority of cases a balance is reestablished—the same kind of accommodation that follows other surgical procedures involving the sacrifice of part or all of an important organ or viscus.

As Doctor Smith points out, we should make an attempt to fit the operation to the case according to the suggestions in his paper. The greatest difficulty is in the cases with very small, highly active glands. In these, one is forced to leave a relatively tiny remnant and one runs the risk of serious impairment of blood supply to this small piece.

In all cases, as individual problems, there is the decision to be made, if one has to miss the ideal, whether one wishes to err on the side of too conservative or too radical a removal of tissue. Here is where one's philosophic point of view affects one's technical procedure. As a rule the conservative appeals to me. A few cases of persistent hyperthyroidism are far easier to handle than a few cases of persistent hypothyroidism. Radiotherapy will straighten out three-quarters of the former, the balance requiring reoperation. In my experience hypothyroidism has usually been permanent, and these individuals have, I firmly believe, a greater quality and quantity of unhappiness than those with persistent hyperthyroidism. People with hypothyroidism are uncomfortable and unhappy. Thyroid extract will of course help to a degree, but it is at best a poor substitute for an adequate supply of one's own thyroid secretion. It is also a misfortune when one has to live with a bottle of pills, from both financial and psychologic reasons.

DR. EMIL GOETSCH (Brooklyn, N. Y.): I would like to say a few words with reference to the remnant remaining after thyroidectomy which Doctor Smith has discussed in detail. In the hyperplastic thyroid gland that has been untreated medically with iodine, there follows, after the intensive preoperative

treatment with iodine, a marked accumulation of colloid, in other words, a marked involution together with a good clinical remission. However, in those instances of hyperplastic thyroid glands which have been treated medically with iodine, perhaps for weeks or months, the preoperative intensive treatment with iodine is not followed by satisfactory involution or by a good clinical remission. Accordingly, one should leave a larger remnant in the case of the colloid gland with a good involution than in the latter instance.

I think it is of the greatest importance to visualize the histologic appearance of the gland that we are resecting, for when we are considering remnants purely in respect to their weights, we may be talking about the weight of the contained colloid that we are leaving behind, which has little to do with the future outcome of the operation. In the remnant, we naturally are planning to leave a certain amount of thyroid parenchyma and if one visualizes the relative amount of parenchyma which one is leaving in the remnant, then I believe we are on a correct basis as to the amount of real thyroid gland which we are leaving for future functional purposes. I believe it is a safe practice to resect the thyroid gland more radically in instances of extreme toxicity in which there is the highest degree of hyperplasia. In other words, the more hyperplastic the gland, the more radically I believe one should proceed. The possibility of recurrence is thus minimized. Recently I saw an instance of recurrence of hyperthyroidism in a woman upon whom I had operated eight years previously, having performed a double resection for exophthalmic goiter. In a review of the history, I found that this woman had an unusually large and extremely vascular gland which was strikingly hyperplastic and of a somewhat beefy, meaty appearance. Following the operation, she developed extremely critical postoperative reaction which was as severe as I had ever seen other than in fatal cases. She fortunately recovered. It would seem that the intense potentiality of developing exophthalmic goiter was not entirely eliminated by the thyroidectomy and that this potentiality was sufficiently strong to develop recurrent hyperthyroidism after eight years.

A further word with reference to the thyroid remnant. Some years ago, during my association with Doctor Halsted, I had occasion to observe the results after single lobectomies, and was surprised to see a rather large number of patients who recovered satisfactorily and apparently remained well following a single lobectomy. In my own earlier experience, before the preoperative use of iodine, I too had performed a considerable number of single lobectomies for exophthalmic goiter in young girls whose further course I was able to observe. Many of these patients subsequently married, had children and remained well over a considerable period of years. These facts indicate that in our concern regarding the size of the remnant to be left behind after thyroid resection, we are considering only one element in the recovery from the very complex disease of exophthalmic goiter. There are many factors concerned other than the size of the remnant and if nature were not on our side as surgeons, we would have a difficult time in the treatment of this disease.

I should like to say a word on one further point and that is with reference to the status of the hyperthyroid patient in the early weeks after thyroidectomy. It is of course dramatic to note a normal metabolic rate and a disappearance of practically all symptoms in the early weeks after thyroidectomy. Such prompt return to normal is often destined to be followed by a late hypothyroidism, two to three or four years or longer, after thyroid resection. In other words, the resection has been too radical and the remnants of thyroid gland remaining after resection too small. In other words, I prefer to leave somewhat larger remnants and see the return to normal following operation occur more gradu-

ally over a period of two to three months, for these patients are destined to remain well and to be more nearly normal than patients who present more normal findings within a few weeks after operation.

DR. MARTIN B. TINKER (Ithaca, N. Y.): Doctor Smith spoke of the weight of the gland, mentioning the size of the fragment left with relation to weight. His experience checks well with our own. As a rule you cannot reduce the size of the fragment remaining to less than 3x1x1 cm., and get satisfactory thyroid secretion. On the other hand, there are, as Doctor Smith has said, cases in which it is necessary, because of low activity, to leave a larger fragment. One of the best ways to arrive at a decision as to how much to leave is to have all interested study the gross specimens in comparison with the microscopic and clinical findings. Any man who will go over 500 specimens in that way, better still 1,000, will get a good idea from the gross appearance of the gland in the neck and the gland when cut, as to how much it is necessary to leave behind. In over 7,000 cases, we have had less than 1 per cent of hypothyroids, also less than 1 per cent, during recent years, of operations for recurrence.

The function of the thyroid depends upon its circulation, and it seems unwise to cut off too much blood supply. We do not ligate the inferior thyroid artery. If radio cutting is used, hemorrhage is controlled without trouble or injury to the artery, and by dividing well up onto the capsule, it is possible to safeguard the parathyroids and enough circulation so that the fragment left will function well and take care of the needs of the patient.

It is very true that in certain instances, a large fragment can be left with satisfactory function. Doctor Schweitzer will remember the cases that Dr. Theodor Kocher operated upon with serious resultant myxedema, where he removed only one lobe, and in my earlier cases, I followed Kocher's technic. Some patients have remained well for 30 years and over, where a single lobe was removed. However, that would not apply to the diffuse goiters that Doctor Smith is talking about. They must have the gland reduced decidedly, best judged after careful study of the gross and microscopic appearance has taught how much to leave and also by maintaining adequate circulation of the stump of gland left.

THE IODINE METABOLISM IN EXOPHTHALMIC GOITER*

GEORGE M. CURTIS, M.D., AND ITALO D. PUPPEL, M.D.

COLUMBUS, OHIO

FROM THE DEPARTMENT OF RESEARCH SURGERY OF THE OHIO STATE UNIVERSITY, COLUMBUS, OHIO

IODINE and thyroid function are inseparably related. The thyroid gland is a principal storehouse for iodine. The thyroid hormone has a high iodine content. The utilization of iodine to form thyroid hormone is thus an integral part of thyroid activity. As a consequence the metabolism of iodine becomes of fundamental significance in the investigation of thyroid physiology, and of the changes in function incident to the development of thyroid disease. The use of iodine in the prevention of goiter has long been known. Since the contributions of Plummer, its use in the preoperative management of exophthalmic goiter has become common clinical knowledge.

Nevertheless, during the past decade newer facts have been added to the iodine story. It is established that iodine is constantly present in human blood. In what form it circulates is not yet clear, although presumably a part, at least, actually exists as thyroid hormone. There is a variable daily loss of iodine in the urine, feces and sweat. The level of the blood iodine fluctuates; likewise the daily excretion in the urine. These findings have gradually assumed clinical significance.

During the past eight years a group of us, including Davis, Cole, Phillips, Barron and Matthews, have investigated various phases of iodine metabolism in over 200 patients with exophthalmic goiter. For determining the minute amounts of iodine present in the blood and urine we have employed three methods: First, an adaptation of the von Fellenberg procedure which was developed by Davis.¹ Second, a similar basic ashing method which was further refined by Phillips.² Third, our present method is a closed, chromic acid oxidation procedure derived by Matthews³ from the Leipert principles, which yields lower values for the blood iodine.

It is difficult to adequately condense the available material. However, further details, together with other tables and charts, are available in current publications.^{4, 5, 6, 7} In the present communication we wish to present four features of the metabolism of iodine in exophthalmic goiter. These concern: (1) the iodine content of the goitrous thyroid gland, which is decreased; (2) the blood iodine, which is usually increased; (3) the urinary excretion of iodine, which is usually increased; and (4) the iodine balance, which reveals a depletion of the patient's usual reserve store of iodine.

In order better to understand the pathologic iodine metabolism which exophthalmic goiter presents, it is clarifying to consider its two principal features. Our studies⁶ have clearly demonstrated a greatly increased mobilization of iodine with a subsequent depletion. This is comparable to the increased

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mobilization of calcium and the ensuing calcium depletion which occurs in hyperparathyroidism.

THE THYROID GLAND IODINE.—*The Thyroid Gland Iodine Is Decreased in Untreated Exophthalmic Goiter:* The normal iodine content of the human thyroid gland varies from 8 to 10 mg. The wet weight concentration varies around 40 mg. per cent, while the dry weight concentration ranges around 200 mg. per cent. It is significant that this iodine is contained principally in the colloid substance. Variations occur, physiologically, seasonally and geographically. Thus, the iodine content is lower during the winter months, and higher in persons living in coastal cities where the iodine intake is greater.

Since the original contributions of Baumann it has been known that the diffuse hyperplastic gland, characteristic of exophthalmic goiter, has a diminished iodine content. This observation has been repeatedly confirmed, and more recently by Lunde. It signifies iodine loss and is directly correlated with the loss of colloid substance from the more cellular, hyperplastic alveoli.

Thyroid iodine depletion may also ensue during the course of severe infectious diseases. It may be produced experimentally by the administration of the thyreotropic hormone.

THE BLOOD IODINE.—*The Blood Iodine Is Usually Increased in Untreated Exophthalmic Goiter:* Extensive clinical investigation, subsequent to the studies of Veil and Sturm, has established the value of the blood iodine as a measure of thyroid function. It is usually increased in patients presenting hyperthyroidism, and decreased in those with hypothyroidism. It has become a clinical aid in recognizing hyperthyroidism. In those unusual instances where it is not increased, it has been shown to have prognostic value. Its clinical significance has consequently become similar to that of the blood sugar in recognizing functional variations of the islets of Langerhans, or of the blood calcium in determining changes in parathyroid activity.

TABLE I
IODINE METABOLISM IN EXOPHTHALMIC GOITER

Case No.	Sex	Age	B.M.R.	Blood Iodine Micrograms %	Urinary Iodine Micrograms Daily	Days Observed
332556.....	F.	17	Plus 18	30.9	169	8
331377.....	F.	18	Plus 47	21.2	65	13
326974.....	F.	32	Plus 32	29.1	106	5
326035.....	M.	34	Plus 75	26.7	196	2
332609.....	F.	35	Plus 95	13.5	159	7
326431.....	F.	35	Plus 50	35.0	111	2
331567.....	M.	38	Plus 50	22.7	105	7
335185.....	M.	39	Plus 44	18.0	146	4
335167.....	M.	48	Plus 53	20.8	310	3
335262.....	F.	51	Plus 109	27.5	133	1
335262.....	F.	51	Plus 93	30.6	230	3
<i>Averages.....</i>			Plus 69	26.0	157	5
<i>Normal Averages.....</i>			Plus 10	12.0	51	18
			Minus			

The majority of patients with untreated exophthalmic goiter present an increased blood iodine (Tables I and II). Results obtained from the earlier methods of analysis yielded higher values for the blood iodine than are now found (Table I). These older methods, however, revealed that the blood iodine averaged more than twice normal in patients with exophthalmic goiter. Newer methods reveal a lower normal blood iodine (Table II). Nevertheless, they also show that it is increased to more than twice normal in exophthalmic goiter. The proportional increase in exophthalmic goiter is similar by either method.

TABLE II
THE IODINE BALANCE IN EXOPHTHALMIC GOITER
As Compared with Normal Individuals and Patients with Nodular Goiter

Type of Goiter	Number of Patients	Total days of Investigation	Average B.M.R. %	Average Blood Iodine Mcg.* %	Average Daily Output				Average Daily Intake Mcg.	Average Daily Balance Mcg.
					Urine Mcg.	Feces Mcg.	Sweat Mcg.	Total Mcg.		
1. None: normal persons	3	24	Minus 7	4.3	51	11	9	71	20	-42
2. Nontoxic nodular.....	2	18	Minus 8	3.0	40	10	10	60	25	-35
3. Toxic nodular.....	2	15	Plus 28	8.5	107	50	13	170	39	-131
4. Exophthalmic.....	3	33	Plus 40	9.0	68	55	15	138	20	-109

* Mcg. denotes a microgram (0.001 mg.).

The nature of this increased blood iodine is not clear. It appears to be principally in the alcohol insoluble fraction, which has been designated "organic." Presumably it represents a greater circulation of the high iodine-containing thyroid hormone or of its metabolic products.

There is no direct parallelism between the blood iodine, the urinary excretion of iodine and the basal metabolic rate in patients with exophthalmic goiter (Table I). As a rule, however, all three are increased. Each of the three, however, dependent upon the phase of the disease, may lie within the normal range. Thus, in a late stage of untreated exophthalmic goiter we would ordinarily expect a resultant pronounced iodine depletion to have occurred. This should have an effect upon the blood and urinary iodine.

Iodine tolerance tests have been applied to the diagnosis of exophthalmic goiter. These depend upon the rate at which the injected iodine is removed from the blood stream, as shown by subsequent blood iodine determinations. The progressive iodine depletion of exophthalmic goiter is a significant factor in the evaluation of these procedures. The depleted tissues and particularly the depleted thyroid gland appear to remove more rapidly the increased circulating iodine.

The prognostic value of a low blood iodine in patients with exophthalmic goiter is brought out by the studies of Perkin and Hurxthal.⁸ It has been our experience that the increased blood iodine usually found in exophthalmic goiter returns to a normal range subsequent to an adequate thyroidectomy. They have shown in addition that these patients present no evidence of recurrence. On the other hand, in those patients with a normal blood iodine pre-

operatively, they even find a postoperative increase, and point out an increased tendency to recurrence.

THE URINARY EXCRETION OF IODINE.—*The Urinary Excretion of Iodine Is Usually Increased in Exophthalmic Goiter:*⁵ Iodine is a normal constituent in human urine. The daily excretion fluctuates, however, and appears to depend principally upon the variable food intake, which is inconstant. When a constant, monotonous food regimen is maintained, the daily urinary loss is more uniform.⁹ The age of the individual appears to be a factor. Variable physiologic states, such as menstruation, have a demonstrable effect. The amount of iodine excreted in the urine varies geographically.¹⁰ It is low in those regions where goiter is endemic, as in central Ohio, where it averages 51 micrograms daily. It is increased in localities which are relatively goiter-free, as in New Orleans, where it averages 117 micrograms daily. These latter observations have a definite bearing upon iodine deficiency as related to the incidence of goiter.

The majority of patients with exophthalmic goiter reveal an increased loss of iodine in the urine (Table I). Thus 13 normal persons excreted from 36 to 78 micrograms daily, and averaged 51.¹⁰ In contrast, 24 patients with exophthalmic goiter lost from 46 to 357 micrograms daily in the urine, and averaged 147, which is approximately three times greater than normal.⁵

It is suspected that this increased urinary loss of iodine originates in an increased breakdown of the high iodine-containing thyroid hormone. However, this may not prove to be the only factor since other tissue iodine may play a part. To be correlated with this are the increased blood iodine and the loss of iodine from the hyperplastic thyroid.

The precise form in which iodine is excreted in the urine has not been determined.¹¹ It does not appear to be in the form of thyroxin, either chemically or biologically, but rather in a more simple compound. Solution of this particular problem is important.

We have elsewhere presented extensive data of the urinary excretion of iodine of normal individuals,¹⁰ of patients with exophthalmic goiter,⁵ and of patients with other thyroid diseases.⁹ From these studies it appears that the urinary iodine is of similar significance in disturbances of thyroid function as is the urinary calcium in parathyroid disease.

Nevertheless, in extending these studies it soon became apparent that the blood or urinary iodine represented but fractions of the entire process of iodine metabolism. The blood iodine, normally less than 1 mg., presented the amount in circulation, although a part of this presumably existed as thyroid hormone or its iodine-containing split products. Another part presumably represented the iodine of nutrition.

Moreover, determinations of the urinary iodine did not present sufficient evidence concerning the intake, utilization or storage of iodine. Therefore, it became increasingly evident that the *iodine balance* should be determined. This meant the institution of carefully controlled hospital conditions for measuring the intake of iodine in the food, water and air, as well as its excretion

in the urine, feces, sweat and expired air. The difference between the determined amount of intake and output would then yield a *balance*. In case storage were occurring, this would be positive. With an excretory loss greater than the intake, it would be negative.

THE IODINE BALANCE.—*Exophthalmic Goiter Presents an Increased Negative Iodine Balance.*⁶ It is of advantage to understand something of the normal variables and fluctuations of the iodine balance, in normal individuals as well as in patients with nontoxic goiter, before reviewing the abnormality presented by exophthalmic goiter. Detailed studies of these basic considerations are presented elsewhere.^{6, 7, 12}

Normal persons maintained on a low iodine intake reveal a low negative iodine balance (Figs. 1 and 2, Table II). They appear to require a certain amount of intake iodine daily to remain in balance. Thus three normal individuals with a decreased intake of 29 micrograms of iodine daily excreted 71, resulting in a daily negative balance of 42 micrograms. Fifty-one micrograms, or 72 per cent of the iodine, was lost in the urine. Eleven micrograms, or 15 per cent, was excreted in the feces, while nine micrograms daily, or 13 per cent, was lost in the sweat (Table II).

It is possible to increase the intake iodine by adding to the diet milk with an increased iodine content. This has been prepared by giving dairy herds feeds containing supplemental iodine.¹³ When maintained on such a diet, containing adequate iodine, normal persons remain in positive balance, and may even store considerable amounts of iodine (Chart 1). Too, this storage may be increased by the addition of potassium iodide to their diet (Chart 1).

During a period of starvation the negative balance is not only maintained, but may even increase somewhat (Chart 2). This further indicates a constant daily requirement of iodine, a part of which is presumably to be used in the formation of the high iodine-containing thyroid hormone.

The iodine balance of patients with nontoxic nodular goiter resembles that of normal persons, with possibly an even greater tendency to storage.⁷ It is quite dissimilar to that of exophthalmic goiter patients, since the increased mobilization of iodine is lacking, as well as the subsequent depletion (Table II).

Two patients with nontoxic nodular goiter, maintained on a low iodine intake over a total period of 18 days, showed an average negative iodine balance which was within physiologic limits (Table II). The intake iodine averaged 25 micrograms daily while the output averaged 60 micrograms, resulting in a daily negative balance of 35 micrograms. The greatest excretion was in the urine, averaging 67 per cent. Seventeen per cent was excreted in the feces and 16 per cent was lost in the sweat.

One patient (Chart 3) even showed a greater retention of iodine than normal persons similarly controlled (Table II). This tendency of patients with nontoxic nodular goiter to store iodine, rather than to excrete it, has been noted by Scheffer and v. Megay.¹⁴

Elsewhere, we have presented extensive data which reveal the great dis-

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turbance of iodine metabolism found in exophthalmic goiter.⁶ The increased mobilization of iodine is shown in the rise of the blood iodine, and by the

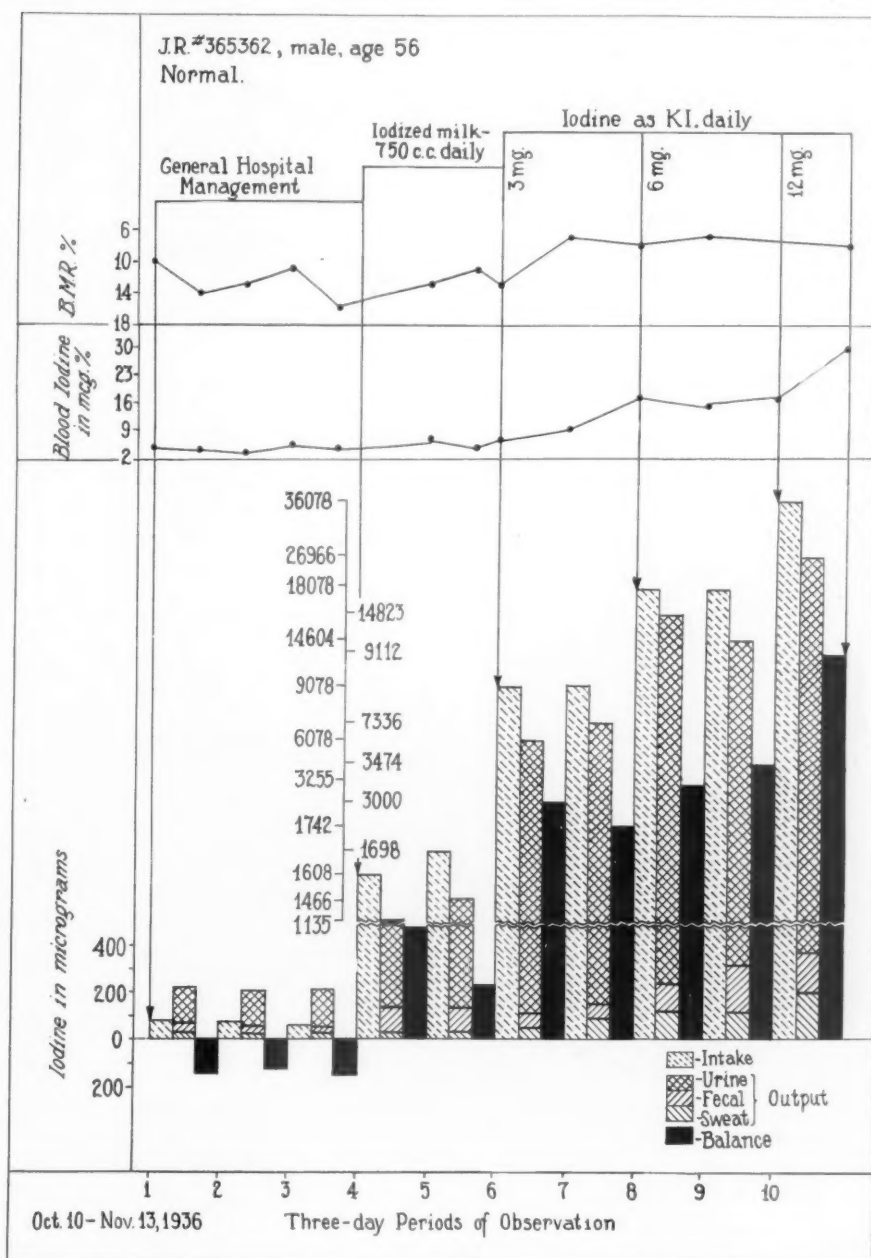


CHART I.—The iodine balance in a normal individual. Note the negative iodine balance on a low iodine intake and the effect of increasing the intake.

greater excretion of iodine in the urine, feces and sweat. Moreover, subsequent to this increased mobilization, iodine depletion ensues. This is revealed

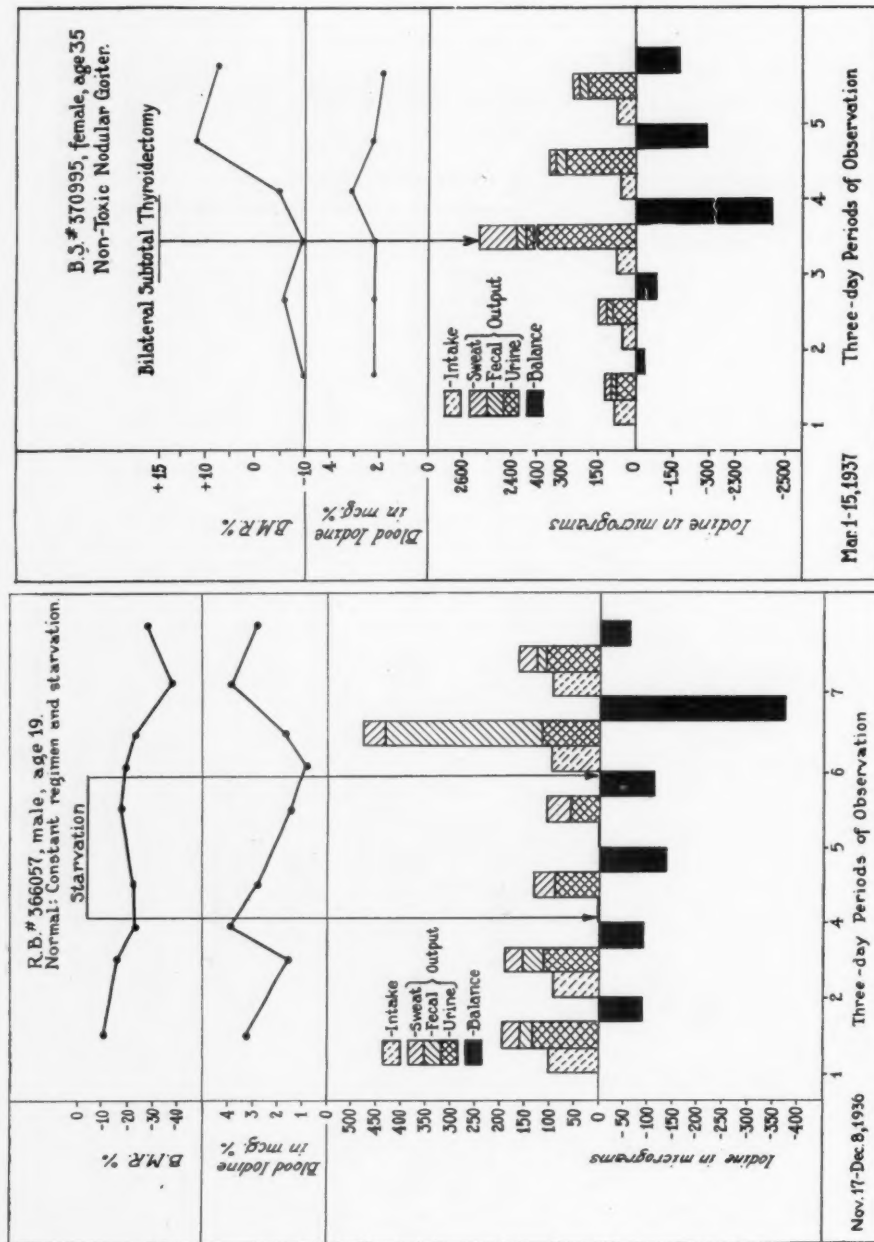


CHART 2.—The effect of starvation on the normal iodine balance. Note the continued negative balance.

CHART 3.—Nontoxic nodular goiter presents a normal negative iodine balance on a low iodine intake. Note the effect of thyroidectomy.

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in the decrease in the thyroid gland iodine and in the greatly increased negative iodine balance.

Patients with exophthalmic goiter, maintained on a low iodine intake, lose from two to three times the amount of iodine lost by normal persons or by patients with nontoxic nodular goiter similarly controlled (Table II). Thus, the intake of three exophthalmic goiter patients averaged also 29 micrograms

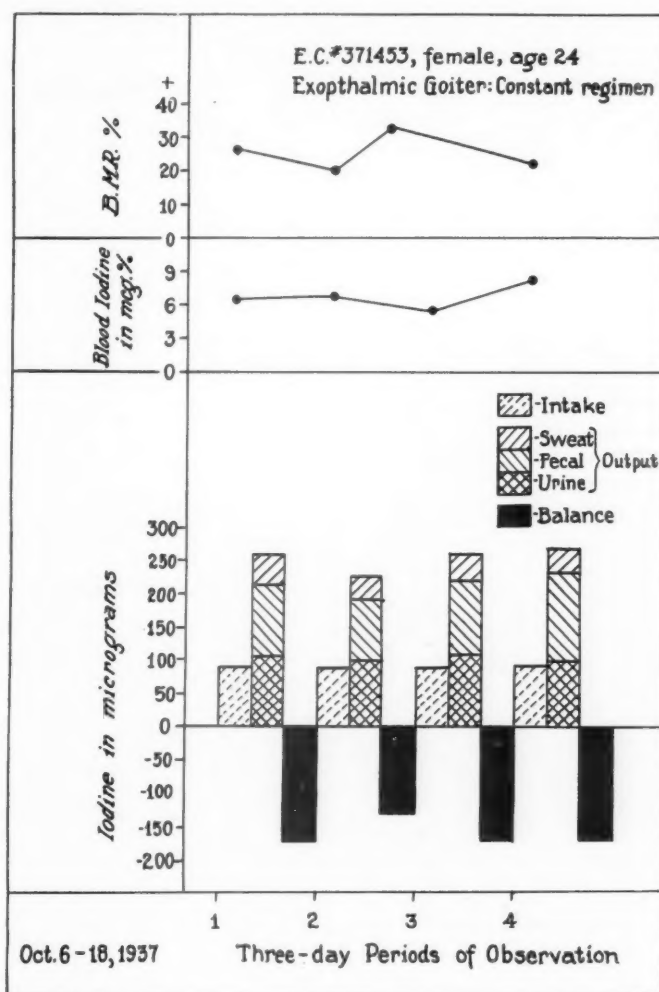


CHART 4.—The increased negative iodine balance of exophthalmic goiter. Note the increased fecal excretion over normal.

daily, while the daily loss was 138 micrograms. This resulted in a daily increased negative iodine balance of 109 micrograms. The greatest excretion of iodine was by way of the urine, averaging 49 per cent. Forty per cent was lost in the feces and 11 per cent in the sweat. The greatly increased fecal loss over normal is shown in Chart 4.

It would consequently appear that untreated exophthalmic goiter is characterized by a tendency to lose iodine. Thus, two patients have been maintained on an iodine intake sufficient to keep a normal individual in positive balance, and to allow for some storage. Both, however, showed a continued negative iodine balance.^{6, 12}

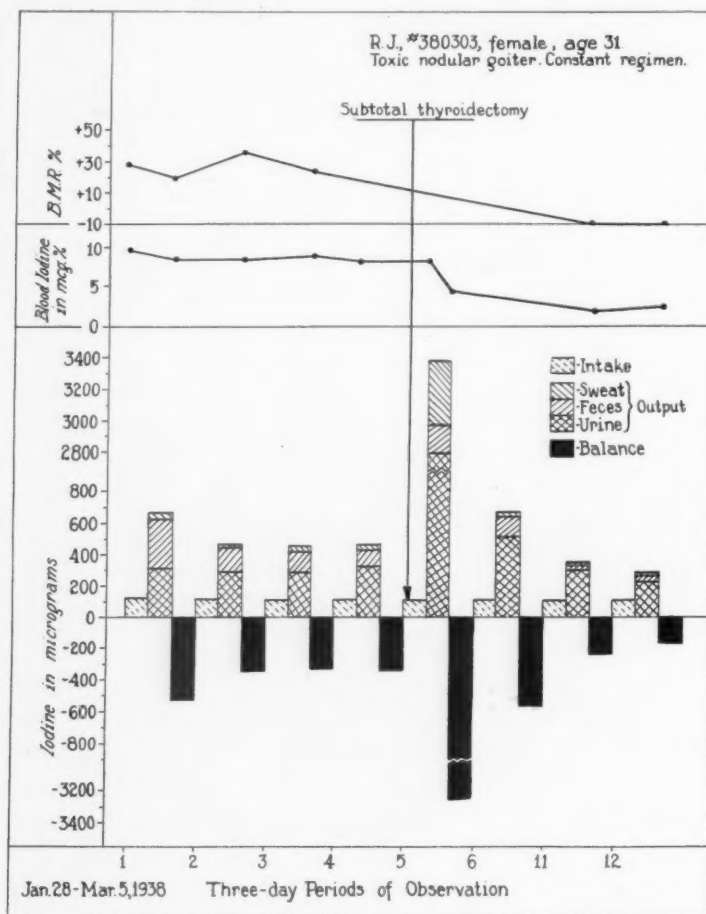


CHART 5.—The increased negative iodine balance of toxic nodular goiter. Note the increased urinary excretion over normal. Note the effect of thyroidectomy.

Nevertheless, *greatly* increasing the intake results in an immediate retention of iodine. Moreover, the resultant positive balance becomes considerably greater than normal (compare Chart 6 of reference 6 with this Chart 1). The diffuse hyperplastic goiter, progressively depleted of iodine during the previous course of the disease, may then rapidly store more than 100 mg. Presumably there is also a lesser repletion of other depleted tissues. This increased storage is maintained for a varying period of time, depending upon the extent to which previous depletion had occurred. However, with the

cessation of the daily administration of 10 mg. of iodine, the former negative balance is soon reestablished, while the stored iodine is then progressively lost.⁶ It appears difficult for a patient with untreated exophthalmic goiter to store or to hold iodine.

The true nature of this increased negative iodine balance of exophthalmic goiter needs further investigation. Other tissue-iodine than that of the thyroid gland may play a part. Too, it is possible that iodine has another function in human metabolism besides furnishing two-thirds of the active thyroid hormone, thyroxin. Presumably, however, the increased iodine loss results from an increased secretion and consumption of thyroid hormone with the consequent greater mobilization and excretion of iodine. This problem has been further discussed elsewhere.⁶

Several factors may influence the increased loss of iodine in exophthalmic goiter. On medical management alone, including hospital control with bed rest, a high caloric diet and calcium therapy, one patient showed a remission of the clinical symptoms, and a decrease of the basal metabolic rate to within normal range. There was a corresponding decrease in the excretion of iodine through the various channels, while the iodine balance returned to within normal limits during the sixth three-day period.⁶

Subsequent to adequate thyroidectomy we have found that the disturbed iodine metabolism of exophthalmic goiter returns to normal.⁶ The blood iodine decreases. There ensues a lessened urinary excretion of iodine, while the fecal and sweat loss are also diminished. The increased negative balance decreases, and eventually comes to lie within the normal range. This may ensue as early as the sixth postoperative day.

In conclusion, we wish to compare the disturbed iodine metabolism of toxic nodular goiter with that of exophthalmic goiter. This will be done briefly, since it has been considered *in extenso* elsewhere.⁷ In both, the blood iodine is usually increased; however, in average more so in exophthalmic goiter.⁴ In both, the urinary iodine is usually increased; however, more so in toxic nodular goiter.⁵ Both present an increased fecal and sweat loss, greater in exophthalmic goiter (Table II). In both, the B.M.R. is usually increased, to a higher range in exophthalmic goiter.

Toxic nodular goiter is thus likewise characterized by an increased negative iodine balance,⁷ which is greater than that of exophthalmic goiter. Too, this also returns to a normal range subsequent to an adequate thyroidectomy (Chart 5).⁷

A summary of our balance studies on ten patients^{6, 7} is presented in Table II. This reveals, by comparison, the increased negative iodine balance of exophthalmic goiter.⁶ However, patients with toxic nodular goiter present an even greater negative iodine balance,⁷ due to a greater urinary excretion.⁵ Nevertheless, the excretion of iodine in the feces and sweat is greater in exophthalmic goiter.⁶ These findings have a direct bearing upon the comparative differences between these two forms of hyperthyroidism.

CONCLUSIONS

Exophthalmic goiter is characterized by an *increased mobilization of iodine*. This is revealed in the elevated blood iodine, and by the increased loss of iodine in the urine, feces and sweat. As a consequence of this increased mobilization, *iodine depletion* ensues. This is demonstrated by the decreased thyroid gland iodine and in the negative iodine balance, which is greatly increased over normal. Exophthalmic goiter thus presents a profound disturbance of iodine metabolism.

There is a striking similarity between the disturbed iodine metabolism of hyperthyroidism and the disturbed calcium metabolism of hyperparathyroidism.

REFERENCES

- ¹ Davis, C. B., and Curtis, George M.: The Quantitative Determination of the Iodine Content of Blood. *Jour. Lab. and Clin. Med.*, **18**, 24, October, 1932.
- ² Phillips, F. J., and Curtis, George M.: The Clinical Determination of Iodine in Blood, Urine and Feces. *Amer. Jour. Clin. Path.*, **4**, 346, July, 1934.
- ³ Matthews, N. L., Curtis, George M., and Brode, W. R.: The Determination of Iodine in Biological Materials. *Ind. Eng. Chem., Anal. Ed.*, in press.
- ⁴ Curtis, George M., Cole, V. V., and Phillips, F. J.: The Blood Iodine in Thyroid Disease. *West. Jour. Surg.*, **42**, 435, August, 1934.
- ⁵ Curtis, George M., and Puppel, I. D.: Increased Urinary Excretion of Iodine in Hyperthyroidism. *Arch. Int. Med.*, **60**, 498, September, 1937.
- ⁶ Puppel, I. D., and Curtis, George M.: The Iodine Balance in Exophthalmic Goiter. *Arch. Path.*, in press.
- ⁷ Puppel, I. D., and Curtis, George M.: The Iodine Balance in Nodular Goiter. *Jour. Clin. Invest.*, in press.
- ⁸ Perkin, H. J., and Hurxthal, L. M.: The Blood Iodine Level Before and After Subtotal Thyroidectomy for Hyperthyroidism. *New England Jour. Med.*, **215**, 698, October, 1936.
- ⁹ Curtis, George M., and Puppel, I. D.: The Urinary Excretion of Iodine in Thyroid Disease. *West. Jour. Surg.*, **45**, 417, August, 1937.
- ¹⁰ Curtis, George M., Puppel, I. D., Cole, V. V., and Matthews, N. L.: The Normal Urinary Iodine of Man. *Jour. Lab. and Clin. Med.*, **22**, 1014, July, 1937.
- ¹¹ Davison, R. A., and Curtis, George M.: Unpublished data.
- ¹² Cole, V. V., and Curtis, George M.: Human Iodine Balance. *Jour. Nutrition*, **10**, 493, 1935.
- ¹³ Phillips, F. J., Erf, Oscar, and Curtis, George M.: The Effects of Prolonged Increased Iodine Feeding. *Ohio Jour. Sci.*, **35**, 286, June, 1935.
- ¹⁴ Scheffer, L., and v. Megay, L.: Jodstoffwechsel bei Kropfträgern. *Klin. Wchnschr.*, **14**, 1360, 1935.

DISCUSSION.—DR. FRANK H. LAHEY (Boston, Mass.): This problem of iodine metabolism is a very interesting one and we have always been interested in Doctor Curtis' investigations. We have carried on some investigations during the past three years in association with Mr. Perkin, a biochemist, and have learned some very interesting and valuable things.

When one realizes the variation in tissue iodine content, it becomes evident, at once, what an important part the thyroid plays in this iodine problem. For instance, Mr. Perkin has made iodine determinations of 10 Gm. of wet tissue and when the 10 Gm. of brain or any of the remainder of the tissue is approximately 23 micrograms, not infrequently there will be 3,500 micrograms of

iodine in the thyroid. That evidences what a part the thyroid plays in iodine metabolism.

We have tried, for a long time, various methods of demonstrating circulating thyroxin, such as the effect of the serum of the patient with hyperthyroidism upon an electrocardiogram of the six, seven, eight or nine day chicken embryo heart, and we have never been able to demonstrate it. We have been interested in blood iodine, of course, as a possible indicator of the amount of thyroxin in the blood stream because 65 per cent of thyroxin is iodine. The iodine fraction is separable and when separated, thyroxin, of course, no longer elevates metabolism.

It has seemed to us, probably in the beginning before we had some of our disappointments, that when a patient had a high blood iodine preoperatively and low postoperatively, which correlates quite accurately with the basal metabolism, that this was probably evidence of the fact that blood iodine is circulating thyroxin, but we have found that 30 per cent of our cases do not have a high preoperative and low postoperative blood iodine. They did always, however, have a high preoperative metabolism. Thirty per cent of our cases have had a blood iodine preoperatively below normal or normal, which postoperatively went above normal and did not come back to normal for six months. Then we found ourselves a little confused.

I would like to present on the other hand, certain interpretations which have proven of great value to us. I would be very much interested to hear from Doctor Curtis what happens to the urinary iodines in these patients who have low blood iodines in the presence of high metabolism.

We have correlated the basal metabolism and blood iodine preoperatively in 110 proven cases of hyperthyroidism, and have charted the course of both of these figures postoperatively at the end of three months and at the end of six months. In these cases, the average basal metabolism was plus 45 and the average blood iodine 22.8 micrograms, normal in this region being 10 micrograms. At the end of three months the basal metabolism had come to normal, the blood iodine to 10 micrograms, and at the end of six months the basal metabolism was at normal and the blood iodine 7.5 micrograms. It is of interest in this group of cases, in which there is the typical preoperative elevation of blood iodine and return to normal correlated with basal metabolism, to note what the percentage of recurrent hyperthyroidism in this group is. We are particularly interested in this because by means of blood iodine we can, with quite definite certainty, establish in what cases recurrence is most likely to occur. In this typical group with preoperatively elevated blood iodines, there is but one-half of 1 per cent recurrent hyperthyroidism. This type of preoperative elevation of blood iodine correlated with basal metabolism both preoperatively and in postoperative drop, represents 70 per cent of all the cases.

On the other hand, in 30 per cent of the cases, there is quite a different picture. The preoperative metabolism is high but the preoperative blood iodine is not only not elevated, but is below normal. At the end of three months the preoperative metabolism has come to normal, but at this time the blood iodine, previously below normal, has now risen to above normal. At the end of six months the metabolism remains normal, and at that time the blood iodine has become normal. It is in this group of cases that one must look for the recurrent hyperthyroidism because 22 per cent of these cases show a recurrence of hyperthyroidism.

When one realizes that our incidence of recurrent hyperthyroidism has been but 3 per cent and that 22 per cent of this group show recurrence, it is

obvious that it is in this group that very radical removals of the thyroid must be performed.

There is another interesting clinical observation in connection with the patients who have low blood iodines and high metabolisms, and that is that in the group having high metabolisms and high blood iodines, but 17 per cent required multiple stages, while in the group having high metabolisms but low blood iodines, 45 per cent required multiple stage operations. It is, therefore, as important to realize that not only does this atypical group represent the patients in whom recurrence is most likely to occur, but also the group in which mortality is most likely to occur and in which cautious operative approach must be undertaken.

Another interesting development which has been demonstrated by Mr. Perkin in connection with blood iodine is that if one makes a scatter chart of patients' blood iodine in relation to the length of time which they have had the disease, it will be found that in a predominating majority of cases, the blood iodine will be elevated above normal when the disease has been present for a year or less but as soon as the disease has been present for a year or more, a predominating majority of the blood iodine determinations will be found to be below normal. This phenomenon is undoubtedly related to exhaustion of the patient's store of body iodine when the disease has existed a sufficient length of time.

As regards Doctor Smith's paper, we think we will probably always perform thyroidectomy more or less by rule of thumb. It will never be possible, I believe, due to the anatomic variations, the way the lobes go behind the trachea and in the groove between the trachea and the esophagus, to make very accurate decisions about the percentage removed.

There are some very valuable points, however, in this connection, that is, how much thyroid to remove in relation to the patient's reaction to iodine. Doctor Cattell, some years ago, reduced 400 thyroids, surgically removed, to a powder and determined the milligrams of iodine per gram of dried gland, correlating this with the histologic picture, that is, the degree of iodine involution. He found that 90 per cent of the thyroid gland would involute and about 10 per cent would not. He found that the very severe cases were those with small thyroids which were very vascular, very soft and did not involute. You can tell pretty well clinically which patient has involuted and which patient has not, and you can tell very definitely at the operating table which patient has involuted and which patient has not.

The patient whose thyroid gland has involuted under iodine will develop firmness in the thyroid gland, and they will show a drop in metabolism, a gain in weight and a drop in pulse rate. The patients who do not show an involution of their thyroid glands do not show these improvements; and at the operating table when you cut the thyroid gland across, the one that is involuted is pale, firm and nonvascular; the one that has not involuted is red, cellular and vascular. It is in the very small, red, vascular cellular gland that has not involuted that radical removals must be performed if one wishes to prevent recurrence of the hyperthyroidism; and it is in the glands that are pale, firm and nonvascular, and the patients who show marked drops in metabolism, gain in weight, drop in pulse rate after the administration of Lugol's solution, that less radical removals of thyroid tissue need be performed.

DR. GEORGE M. CURTIS (closing): We have also observed low blood iodines in patients with exophthalmic goiter; however, our incidence is not so high as that which Doctor Lahey reports. Too, we have noted a low

urinary excretion of iodine in certain patients with exophthalmic goiter. That is also unusual; I should estimate less than 20 per cent.

Doctor Lahey's "scatter chart" has shown a general decrease in the blood iodine as the disease progresses. On the basis of our studies, a part of which have been presented here, this might have been predicted, since the increased mobilization of iodine in exophthalmic goiter eventually leads to iodine depletion. The increased mobilization is shown in the increased blood iodine, and in the greater than normal iodine loss in the urine, feces and sweat. The resultant iodine depletion is demonstrated by the decreased thyroid gland iodine and particularly by the increased negative iodine balance. Patients with exophthalmic goiter thus progressively deplete themselves of iodine in a similar manner as patients with hyperparathyroidism deplete themselves of calcium.

If we could visualize the onset of exophthalmic goiter, it would appear to commence in a normal thyroid gland with a normal iodine content. Precisely what institutes the hyperplasia or what causes the alveoli progressively to lose colloid and consequently iodine, is not clear. Nevertheless, as the disease continues, depletion ensues and increases. Since Baumann's observations, in 1895, it has been known that the iodine store of the diffuse hyperplastic goiter becomes diminished.

By hypothesis, the "scatter chart" appears to present a similar story of progressive iodine depletion as reflected in the blood iodine. We would expect a similar change in the urinary excretion. The severity of the disease and its duration both modify the variable amount of iodine depletion. If this is severe and if resultant damage has followed, it may explain the tendency of patients with low blood iodines to have recurrence.

MEDIASTINITIS FOLLOWING CERVICAL SUPPURATION

HERMAN E. PEARSE, JR., M.D.

ROCHESTER, N. Y.

FROM THE DEPARTMENT OF SURGERY, THE UNIVERSITY OF ROCHESTER, SCHOOL OF MEDICINE AND DENTISTRY,
ROCHESTER, N. Y.

INFECTION of the mediastinum may originate from so many different sources and have such divergent manifestations that the term "mediastinitis" means little unless qualified by a description of its type and kind. The process may range from a simple, nonsuppurative inflammation in association with pericarditis, bronchitis, influenza or pneumonia to a very grave, often lethal, diffuse suppurative phlegmon. A chronic variety is seen in tuberculosis which is sometimes called mediastinopericarditis. Tuberculosis also involves the tracheobronchial lymph nodes with occasional suppuration to form a tuberculous mediastinal abscess. These same nodes are infected in many upper respiratory infections, and should they suppurate, a pyogenic mediastinal abscess results. This phase of the subject has recently been emphasized by Lerche.³⁵ Pyogenic abscesses also follow invasion of the mediastinum from contiguous lung abscesses, empyema, cervical infection, spondylitis, perforating wounds or retroperitoneal infection. These abscesses usually develop slowly enough to allow time for diagnosis, localization and drainage. They have been cured by spontaneous rupture into the trachea or esophagus, repeated aspirations by needle puncture, dorsal mediastinotomy, sternal trephine or cervical drainage. In contrast, a mediastinal phlegmon spreading through so vital a spot may be quickly lethal unless strenuous efforts are undertaken for its control. This diffuse suppuration of the mediastinum may come from any of the sources causing localized abscess if the speed and magnitude of the contamination is sufficient; but the visceral perforations of chest and neck are its commonest cause, and of these the cervical lesions are the most frequent. The consideration of diffuse suppurative mediastinitis might well begin with a study of infections in the neck that gravitate into the mediastinum. This demands a knowledge of the fascial spaces connecting the two, for the infection travels along these and it is in them that the surgeon must intercept or drain it. The spaces lie between layers of the cervical fascia, a structure that is so complex that if followed through all of its ramifications is apt to resemble a maze. The subject may be greatly simplified by considering only that part of the cervical fascia and its spaces which significantly relates to the spread of infection. For this purpose it is sufficient to deal with the viscerovascular compartment which contains the visceral space in the center and around it, the prevascular space, the retrovascular space and the vascular sheath on either side.

The first experiments designed to study the compartments of the neck were undertaken by Bichat.² But many years elapsed before an intensive

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investigation by injection methods was made by Henke,²¹ König and Riedel,²⁷ Soltmann,⁶¹ Poulsen⁵⁴ and Schmitt.⁶⁰ During this period, from 1872 to 1893, most of the essential facts were obtained about the cervical fascia, and the manner of dependent spread of infection along its spaces. For no apparent reason, much of this information has not been referred to in the current literature and references to it are frequently lacking. Some years ago, personal interest in the matter led to anatomic and postmortem dissections in order to study the paths of dependent spread of cervical infection. In doing these the articles of Mosher,⁴⁴ Furstenberg¹⁰ and Iglauer²³ were helpful. Recently Collier and Yglesias⁷ have reported anatomic studies on this subject.

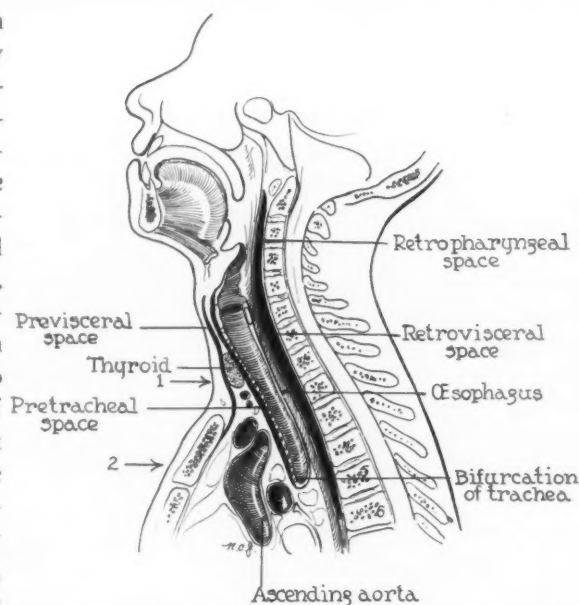


FIG. 1.—A longitudinal section to show the cervical spaces. In front is the previsceral space which ends at the sternum and does not enter the mediastinum. Next is the pretracheal space which conveys infection from tracheal and thyroid gland operations. Behind is the retrovisceral space, the route traveled by pus in 71 per cent of cases of mediastinitis from cervical suppuration. Note that the retropharyngeal space is not separated from it but is only its upper portion. Numerals indicate the level of cross-section, for Figures 2 and 3 of the text.

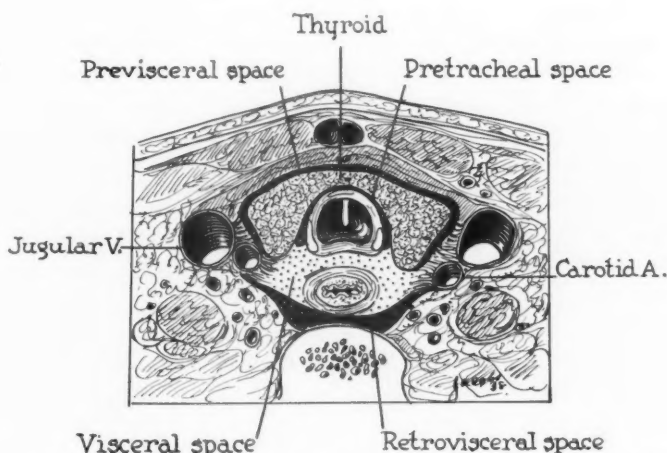


FIG. 2.—A cross-section at the level of the thyroid gland. The visceral space including esophagus, trachea and thyroid gland is a compartment surrounded by the pretracheal fascia in front and the buccopharyngeal fascia behind. In its pretracheal portion is a true space. Behind is the retrovisceral space. Note its relation to the esophagus and cervical spine.

The following summary is an attempt to appraise the facts obtained from the literature in the light of clinical observation and anatomic dissection.

THE VISCEROVASCULAR COMPARTMENT.—That part of the neck occupied by the pharynx, larynx, trachea, esophagus, thyroid and thymus glands, nerves and great vessels is often termed the viscerovascular compartment. It

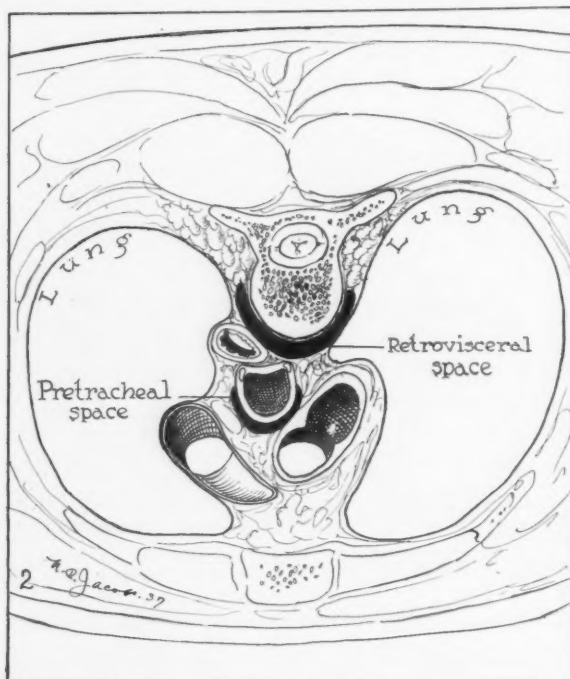


FIG. 3.—Section in the chest at the level of the fifth dorsal vertebra. The retrovisceral space is in close relation to the pleura.

extends from the base of the skull to the mediastinum where it ends by fusion of its fascial structures about the bifurcation of the trachea, the aorta, innominate vein and pericardium. The consideration of this area as a compartment is for the purpose of orientation, since only in its subdivisions are found actual or potential open spaces. These are shown in longitudinal section in Figure 1, and in cross-section, at two levels, in Figures 2 and 3. Reference to these figures will help to clarify the following description.

Visceral Space.—The area bounded by the pre-

tracheal fascia in front, the vascular sheath laterally, and the buccopharyngeal fascia behind, which contains the trachea, esophagus, thyroid gland and nerves, is called the visceral space. It is more potential than real, even though its fascial envelope is one continuous layer. The pretracheal fascia which forms its anterior boundary extends from the hyoid bone to the pericardium, splits to enclose the thyroid gland, then merges laterally with the carotid sheath. The same layer is continued behind the pharynx and esophagus as the buccopharyngeal fascia.

In the anterior portion of this compartment, between the posterior leaf of the pretracheal fascia and the trachea, is a free space, often called the pretracheal space, which extends from the larynx to the bifurcation of the trachea. It has no connection with the spaces in the floor of the mouth or those about the pharynx, so does not convey infection from them. The pretracheal space is usually open during the course of a thyroidectomy, and should infection follow the operation, it may gravitate into the mediastinum through this channel. In performing a tracheotomy the space would seem to be contaminated, yet

mediastinitis from this source rarely occurs. Perforating wounds of the larynx and trachea or operations upon these structures may open and contaminate the pretracheal space, with resultant gravitation of infection into the chest.

In Figures 1 and 3 it is seen that the mediastinal portion of the pretracheal space lies behind the great vessels between the arch of the aorta and the trachea. It is apparent that a surgical approach through the sternum would be difficult because of the interposition of these vessels. The approach through the neck entering the space in the suprasternal notch, below the thyroid isthmus, permits its drainage with the least manipulation. Should the mediastinitis follow thyroidectomy, tracheotomy or other operative procedures in this region, then immediate opening of the wound for drainage should be done.

Infection in the visceral space outside of its pretracheal compartment rarely leads to mediastinitis. The fibrous attachments about the vascular sheath, between trachea and esophagus and between the buccopharyngeal fascia and esophagus impede gravitation of the infection. The usual result is a localized abscess. This is most often seen in perforations of the anterior or lateral walls of the esophagus where, instead of a diffuse spreading infection, one finds a localized suppuration requiring only drainage for cure.

The Previsceral Space.—This compartment is familiar to surgeons as the space used in freeing the thyroid gland at operation. It lies beneath the strap muscles and in front of the pretracheal fascia and thyroid gland, extending from the attachment of the sternothyroideus on the thyroid cartilage and trachea down to the manubrium. Furstenberg¹⁰ has emphasized the importance of a process of the pretracheal fascia which attaches to the posterior surface of the sternum and effectively blocks the lower end of the previsceral space. This important attachment prevents infection from reaching the mediastinum.

Carotid Sheath.—There is a difference of opinion as to the importance of this structure in conveying infection into the mediastinum, for on the one hand Mosher⁴⁴ says: "the carotid sheath . . . is the natural highway for pus and for the surgeon in pursuit of pus"; while Parsons⁵¹ does not think the sheath exists until it is "manufactured . . . with the scalpel." It is immaterial whether the pus is considered to run down a closed sheath or to burrow along the loose areolar tissue beside the vessels, for in either event the vessels act as a guide for its descent. The infection may arise from a suppurative adenitis of the deep chain of lymphatics in this region or from a suppurative thrombophlebitis of the internal jugular vein. An equally important source is from inflammation in the parapharyngeal space, a triangular cone-shaped compartment with its base at the skull and its apex ending around the carotid artery (Fig. 4). This space may be contaminated from a needle puncture in performing a tonsillectomy under local anesthesia, or tooth extraction with nerve block, or it may be invaded from a dental abscess in the second or third molar. From Figure 5 it is apparent how a parotid, peritonsillar or retropharyngeal abscess may rupture into it. Any of these causes of parapharyngeal

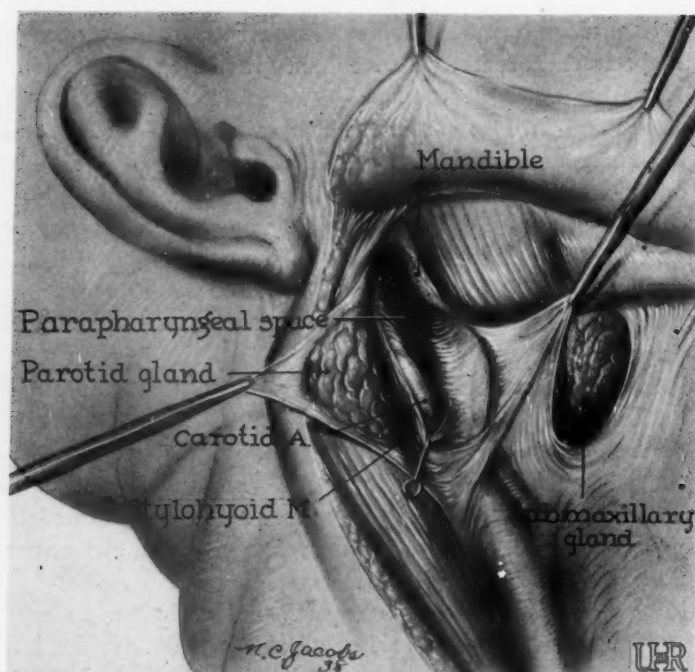


FIG. 4.—The parapharyngeal space seen from the outside. The fused fascia is left in front to separate it from the submaxillary space. The parotid gland is turned back in this dissection for exposure. This could not be done so widely at operation without facial nerve injury. The parapharyngeal space extends up behind the angle of the jaw and ends below around the carotid artery.

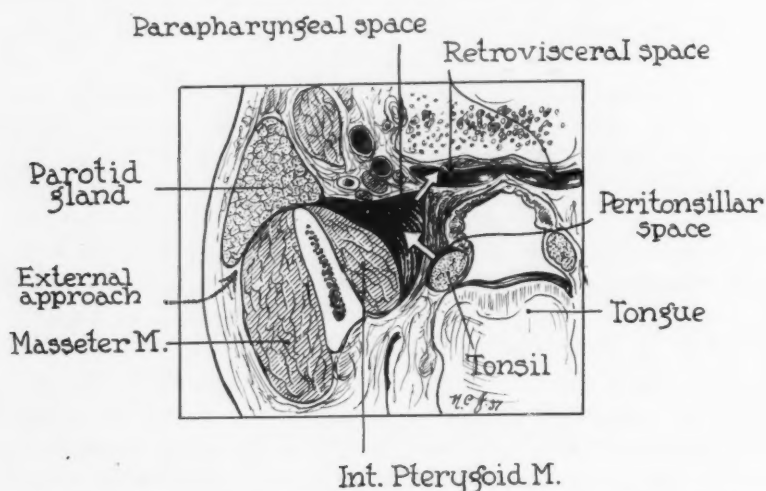


FIG. 5.—The parapharyngeal space may be invaded from a tonsillar, parotid or retropharyngeal infection. Pus from this space may track down the carotid sheath or rupture into the retrovisceral space to involve the mediastinum. (This figure is reprinted through the courtesy of the Journal of the Missouri State Medical Association.)

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space infection may result in mediastinitis by gravitation of pus down along the great vessels. Should this occur, warning is given by a rise in temperature and pulse and by tenderness in the neck.

Another pathway from the parapharyngeal space to the mediastinum is by rupture into the retrovisceral space. When this occurs it may be difficult to detect, as was learned to our sorrow in one patient with peritonsillar, parapharyngeal infection who developed mediastinitis without showing any signs in the neck.

Gravitation of pus along the carotid sheath to invade the mediastinum may occur very rarely in Ludwig's angina. This infection involves the sublingual-submaxillary space (Fig. 6) which is closed so completely by muscle or fascia

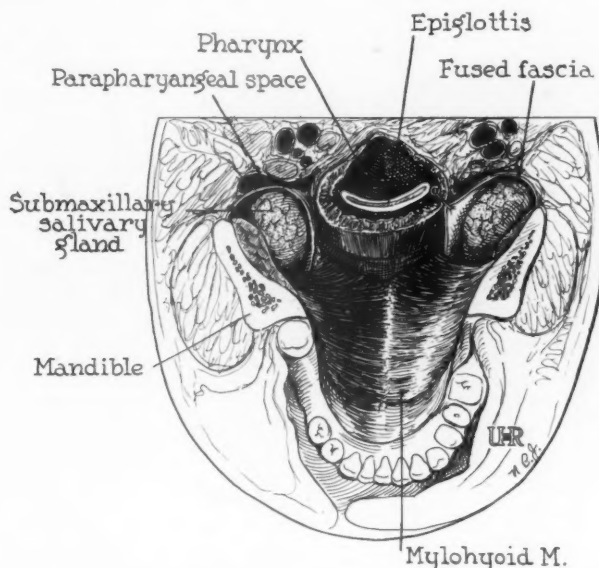


FIG. 6.—The sublingual-submaxillary space, which is involved in Ludwig's angina, is shown by a section through the mouth with the tongue removed. It is separated from the parapharyngeal space behind by the fused fascia which must be eroded through before the infection can gravitate down the neck by this channel.

that no preformed avenue of escape exists. In late, neglected cases the fused fascia may be eroded, allowing drainage of pus into the parapharyngeal space and so down the great vessels or, as was shown by Poulsen,⁵⁴ the infection may rupture out along the facial vessels and follow them to the carotid sheath. Both of these possibilities are remote, and in Ludwig's angina the toxemia from infection or respiratory obstruction is more to be feared than mediastinitis.

Retrovisceral Space.—The space behind the pharynx and esophagus is the most important highway of communication between the neck and chest, for it is the route traveled by infection in 70 per cent of the cases. It is bounded by the buccopharyngeal fascia in front, the prevertebral fascia behind, and the carotid sheaths laterally, and extends from the base of the skull to the bifurcation of the trachea. Its lower limit is usually at the level of the sixth dorsal

vertebra, where it is closed by the fibrous tissue about the tracheal bifurcation. Below this obliterated point the space continues to the diaphragm, but this part is not involved in cervical infection. The thin layer of the buccopharyngeal fascia is the only structure separating the pharynx and esophagus from this cavity, therefore, perforation of the posterior wall of these viscera permits direct contamination of the space. This initiates a most rapidly progressive form of mediastinitis, for repeated swallowing forces food, fluid, air and bacteria through the perforation, which mechanically distends the retrovisceral space from top to bottom. In

a matter of hours, it may be filled with infected material. The extravasated air can be demonstrated roentgenologically, as is shown in Figure 7, which is a lateral roentgenogram of the neck in a patient with an esophageal perforation.



FIG. 7.—The retrovisceral space is defined by air which has escaped into it through a perforation of the esophagus. The upper arrow shows the retropharyngeal part of the cavity while the lower arrow points to a diffuse emphysema in the space.

The retrovisceral space may also be contaminated by an osteomyelitis or tuberculosis of the cervical vertebrae, in which the infection has eroded through the prevertebral fascia. It has been stated that pus in the parapharyngeal space occasionally ruptures into the retrovisceral space rather than following its usual course down the carotid sheath. Another cause of involvement is from gravitation of a retropharyngeal abscess. The retropharyngeal space is only the upper part of the longer retrovisceral compartment and has no true separation from it.

At first glance, one might wonder why retropharyngeal abscesses remain so localized in this free space, but when it is recalled that they begin as a suppurative lymphadenitis, it is apparent that the slow development of the inflammation allows it to seal off from the lower part (Fig. 8). Should this obstruction weaken, there is nothing to prevent the infection from sinking into the mediastinum.

INCIDENCE OF SUPPURATIVE MEDIASTINITIS.—The foregoing discussion deals with those cervical infections that may cause mediastinitis and the paths they may take to get into the chest. The question arises, how frequently does this happen? Hare¹⁶ states that: In 520 cases of mediastinal lesions, consisting of benign or malignant tumors, lymphomata, cysts and infections, there were 78 cases of pyogenic suppuration. The majority were from traumatic wounds penetrating the chest or from intrathoracic suppuration and only 17 came from the descent of cervical infection. Using these statistics

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as criteria, we find that pyogenic suppuration causes 15 per cent of mediastinal lesions, and that 22 per cent of these infections gravitated from the neck.

The question next arises as to what cervical infections are most apt to produce mediastinitis and what is the relative incidence of each in its causation. In an attempt to answer this, 110 cases of suppurative mediastinitis following infection of the neck have been studied; of these, 99 were obtained from the literature* and 11 from personal experience. They are grouped in order of relative frequency in Table I.

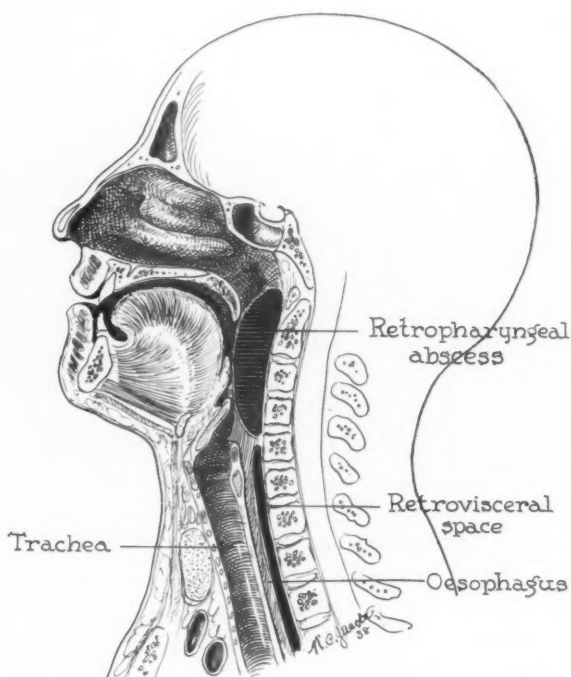


FIG. 8.—A retropharyngeal abscess occurs in the upper part of the retrovisceral space, but the slow development of the supuration allows walling off of the pus and a separation into two compartments. Should this barrier break, there is nothing to prevent gravitation into the chest.

The data in Table I may be regrouped to show the relative importance of the different paths of spread of pus from the neck to the chest. The retrovisceral space conveys infection from perforation of the esophagus, retropharyngeal abscess and spondylitis of the cervical spine. The carotid sheath conducts it in most instances of suppurative lymphadenitis, peritonsillar abscess and Ludwig's angina. The pretracheal space is the course followed in infection after tracheotomy or thyroidectomy. Listed in this way we find them involved as follows: Retrovisceral space: 78 cases, or 71 per cent;

* All pertinent articles listed under mediastinum in Quarterly Cumulative Index Medicus were searched for cases. It is probable that this list is incomplete, since some may have been published under titles that would not be filled under this classification. The total number is small but perhaps is sufficient to indicate trends.

carotid sheath: 23 cases, or 21 per cent; pretracheal space: 9 cases, or 8 per cent.

TABLE I
110 CASES OF SUPPURATIVE MEDIASTITIS

Etiologic Factor	No. of Cases	Per Cent	Operative Result		Nonoperative Result	
			Recovery	Death	Recovery	Death
Perforation cervical esophagus	64	58.1	24	9	4	27
Suppurative cervical lymphadenitis	13	11.8	7	6		
Retropharyngeal abscess.	11	10.	6	3	1	1
Peritonsillar abscess.....	8	7.2	2	2		4
Tracheotomy.....	6	5.5	1	1	1	3
Spondylitis cervical spine	3	2.8	2			1
Postoperative thyroidec-tomy.....	3	2.8	1	2		
Ludwig's angina.....	2	1.8	1	1		
Total.....	110		44	24	6	36

In a majority of the cases studied the infection came from perforation of the cervical esophagus. Neuhof,⁴⁷ in a report on mediastinal suppuration from both cervical and thoracic sources, found perforation of the esophagus the cause in "almost 50 per cent of the cases." This viscus may be penetrated by external traumatic or surgical wounds, or perforated from within by foreign bodies, bougies, an esophagoscope, or eroded by tumors. The author and Doctor Heatly,¹⁸ in a study on the management of esophageal perforation, decided that immediate external operation is the treatment of choice in order to prevent the occurrence of mediastinal infection and the consequent necessity for its drainage. This view is strengthened by the results of treatment of suppurative mediastinitis shown in Table I. In the 64 cases of perforation of the cervical esophagus, 33 received early operation with nine deaths, a mortality of 27 per cent; while in the 31 cases not operated upon, 27 died, a mortality of 87 per cent.

THE PREVENTION OF MEDIASTITIS.—The mediastinitis which follows cervical suppuration results from a dependent spread of infection along the fascial planes. If this gravitation of pus could be blocked, the chest infection would be prevented. Reasoning along these lines, von Hacker¹⁵ proposed a prophylactic operation for packing the spaces in the neck. This procedure was popularized by Marschik,³⁹ and has been described by Palmer⁵⁰ and Glogau¹³ in this country. Recognition of the importance of the retrovisceral space as a path for the descent of pus led to the development of an operation to block it.⁵² These procedures seek to erect a transverse barrier of adhesions across the fascial spaces that connect the neck with the chest. They are indicated in the traumatic visceral perforations, especially of the esophagus

where the progress of the infection is very rapid. Here, even if the surgeon is too late to interrupt the gravitation, he can drain the space and, by releasing tension, prevent extension to the chest.

There is much less indication for prophylactic block of the fascial spaces in the absence of rapidly spreading infection from a visceral perforation. Most suppurations in the submaxillary-submental, parapharyngeal and retropharyngeal spaces or in the carotid sheath are best attacked directly and drained. If the pus has descended in the neck, it will have done so slowly and should be released without destroying the inflammatory adhesions below it. Dissemination of infection might well follow an attempt to do more than this.

MANAGEMENT OF MEDIASTINITIS.—There is every reason to believe that suppuration in the mediastinum should be attacked surgically and drained just as infection in a more accessible location would be. This principle is applicable irrespective of the source of the pus. In the 110 infections which came from the neck, 68 were operated upon with 24 deaths, a mortality of 35 per cent, in contrast to an 85 per cent mortality when operation was not performed. The drainage was accomplished through the esophagoscope in 13, the chest wall in 14 and the neck in 41. There is some controversy among endoscopists as to whether infection should be drained through the endoscope or by external incision. It is probable that the endoscopic approach should be limited to the evacuation of a localized abscess that impinges on the esophagus or presents behind the pharynx.

Incision through the chest wall may be by the anterior or posterior approach. The former is rarely necessary for drainage of infection but the posterior route is useful. The technic of dorsal mediastinotomy is described by Lilienthal,³⁶ while its historical background is given by Gaudiani.¹² It is the operation of choice in draining suppuration below the level of the sixth dorsal vertebra, and is often advisable in evacuating chronic abscesses of the posterior mediastinum at any level in order to collapse their walls. It is a more formidable procedure than the operation through the neck, which should be used whenever possible.

Cervical mediastinotomy was described by von Hacker,¹⁵ though Lurman³⁷ reported, in 1876, drainage of a mediastinal abscess that presented in the neck. This operation is the logical approach for drainage of mediastinitis above the level of the sixth dorsal vertebra. That which originates from cervical infection falls in this category and should be so treated, for as Furstenberg¹⁰ states: "To drain an infection through the tissues which it has invaded, is, I believe, a surgical axiom." It has the advantage of allowing direct inspection of the extent and location of the suppuration.

The incision in the neck is usually made parallel to the lower, medial border of the sternocleidomastoid muscle, though it may be placed transversely to follow the skin folds. The sternocleidomastoid is retracted and the fascia lateral to the sternothyroid muscle is divided to expose the carotid sheath and

thyroid gland. The lateral, and perhaps the inferior thyroid, veins are ligated and divided. This allows lateral retraction of the vessels and medial displacement of the thyroid gland, in order to expose the trachea and esophagus. If a short inferior thyroid artery prevents this, it is ligated and divided. At this stage the carotid sheath and pretracheal space may be inspected, but if uninvolved, they are not opened and the dissection is carried behind the esophagus. This opens the retrovisceral space as is shown in Figure 9. If pus is encoun-

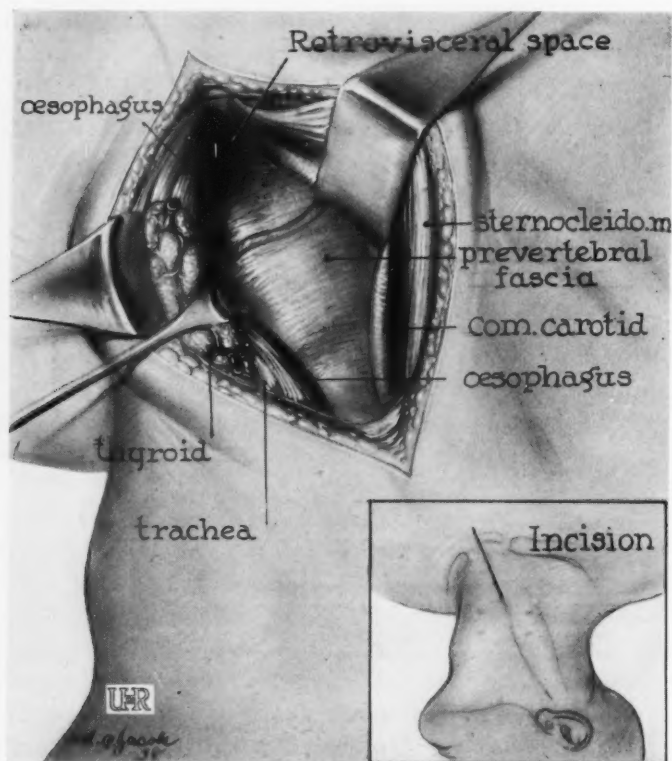


FIG. 9.—Looking down into the posterior mediastinum through the retrovisceral space, as it is seen at operation. Orientation is easier if the drawing is turned so the head is up. The thyroid gland, trachea and esophagus have been retracted mesially, while the carotid artery, jugular vein and sternocleidomastoid muscle are displaced laterally. This exposure permits visual inspection of the space.

tered, it is aspirated and drains are placed to the bottom of the cavity. Some surgeons prefer the approach behind the sternocleidomastoid muscle, but danger of nerve injury is greater in this location. The important factor is to obtain direct drainage, for failure to do this may allow pocketing and residual abscess formation. If the infection has dependent pockets on either side of the midline, they should be drained through separate incisions on the corresponding sides of the neck. A drain crossing the midline is pinched between the spine and the esophagus and causes obstruction to the drainage and abscess formation as is illustrated in Case I.

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Case 1.—Hosp. No. 85308: A female, age 45, was seen 30 hours after an instrumental perforation of the esophagus. The pain, swelling and emphysema were most marked on the left side of the neck, so the incision was made there. But it was found that dependent pockets were present in the mediastinum on both sides, that on the right being the deeper of the two. The patient's condition was very poor, so drains were inserted into both of these cavities and brought out through the incision on the left side. The plan was to perform a second operation on the right side but the patient was too ill to warrant this procedure. She was comatose and aroused only in delirium. The tempera-

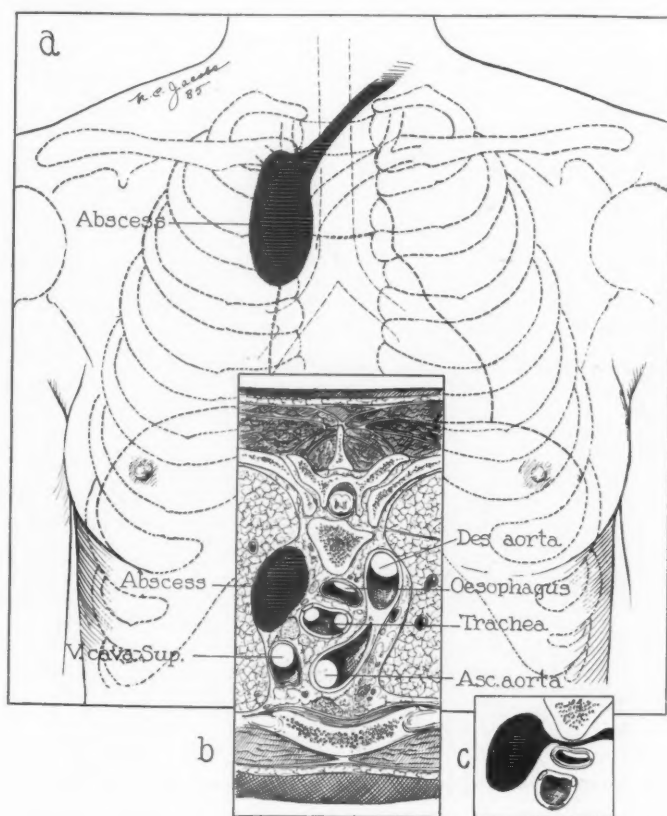


FIG. 10.—Case 1: Residual mediastinal abscess three months after an acute mediastinitis from esophageal perforation. The location in the vertical plane is shown in (a); and in horizontal section in (b). The pinching of the tract between the spine and esophagus is shown in (c). This caused incomplete drainage of the suppuration and allowed the abscess to persist.

ture, pulse and respiration were sustained at a high level. Cyanosis was so marked that an oxygen tent was required constantly. Subcutaneous saline, intravenous glucose, and transfusions were administered, to maintain the fluid balance, while a gastrostomy was performed for feeding. For nine days she held on to life by a narrow margin and then improved rapidly. The fever subsided, mental clarity and physical strength returned and she appeared to be on the road to recovery. Whereas, before she was too sick to drain through the right side, it was now thought that she was too well to need it. This was wrong, for drainage persisted. Injection of the tract showed a residual abscess in the mediastinum. Prolonged attempts were made to drain this by suction through a catheter, but the abscess persisted. Finally, three months after the operation for the mediastinitis, the abscess was drained through the right side of the neck. The patient died six days

after operation of bronchopneumonia, having survived the acute phase of her illness but succumbing to one of its sequelae.

Autopsy revealed a thick-walled abscess cavity, free of pus, situated in the right posterior mediastinum at the level of the aortic arch (Fig. 10). The old drainage tract was compressed between the vertebra and esophagus. The left side of the mediastinum which had received direct drainage was completely healed.

It may be, that it is always more advisable to drain through an incision on the right side, but now after the experience in Case 1, if bilateral pockets are found to exist, both sides of the neck are opened. The exploratory incision should be made on the right side, as anatomic relations make this preferable.

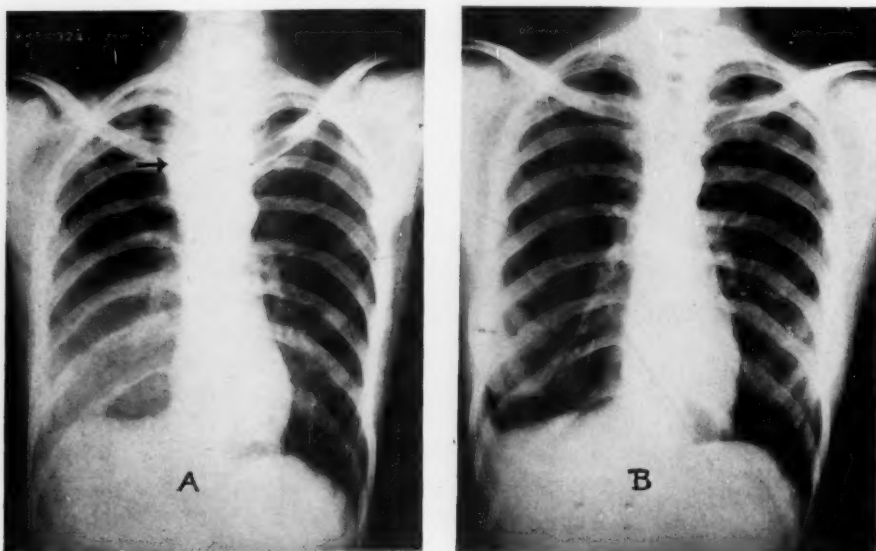


FIG. 11.—Case 2: (A) The arrows point to a residual abscess which has formed during recovery from acute mediastinitis. The pleural edema at the right base is still present. (B) The abscess has been drained and the chest is clear.

The postoperative care of patients with mediastinitis is important, for they are sick and need much supportive treatment. Immediately after operation they should be placed in the Trendelenburg position and kept there until the drainage diminishes or ceases, as otherwise the pus will not run out of the dependent pocket. This position becomes tiresome, but if not maintained may lead to a residual abscess, as happened in Case 2.

Case 2.—Hosp. No. 56324: A female, age 20, lacerated the cervical esophagus with a clam shell. She was operated upon 48 hours after the perforation. Pus was found in the posterior mediastinum. Drainage was established through the incision on the right side of the neck. A gastrostomy was performed the following day for feeding. Fluids were forced by subcutaneous and intravenous infusions. She ran a septic type of temperature, pulse and respiration for one week and then, as these were subsiding, the head of the bed was elevated slightly. Following this the drainage diminished, and the septic phenomena returned. A residual mediastinal abscess (Fig. 11) was demonstrated on the 13th postoperative day, which was drained by inserting a catheter into it

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through the wound in the neck. She was returned to the Trendelenburg position, which, combined with aspiration and irrigation through the catheter, resulted in the evacuation of much pus. Her septic course promptly subsided. Drainage ceased at the end of two weeks, and the catheter and gastrostomy tube were removed; feeding by mouth was resumed, and the wound in the neck was allowed to heal. Recovery was uneventful.

This case illustrates the necessity of keeping the patient's head down, in order to provide dependent drainage until the discharge diminishes or ceases.

The mediastinitis which comes from perforation of the pharynx or esophagus may be complicated by the feeding problem. The swallowing of fluids or food, in the presence of an opening in the tract, allows additional extravasation and contamination and should be eliminated. Absolute rest of the esophagus is impossible, for patients will always swallow some saliva. Relative inactivity, however, may be accomplished by employing an indwelling stomach catheter or establishing a gastrostomy. The tube feeding would appear to be the simpler, but it has been tried repeatedly and found to be less satisfactory than a gastrostomy. Artificial feeding is continued until drainage from the mediastinum stops, then liquids and soft solids are tried by mouth before removing the gastrostomy tube. Should an esophageal fistula be suspected, a drink of dilute methylene blue solution will confirm its presence by its appearance on the dressings of the wound in the neck. No difficulty has been experienced with persistent fistula or stricture formation in these traumatic perforations of the esophagus.

The maintenance of an adequate fluid balance is accomplished by the administration of Ringer's solution or glucose given subcutaneously, intravenously or by rectum. Blood transfusion is administered when indicated by the presence of anemia or depletion of serum protein, for either of these may occur in patients who are receiving a low protein intake in the presence of a suppurative infection. Another measure of value is the liberal use of oxygen in the immediate postoperative period, as cyanosis is often present in suppuration of the mediastinum. Some patients show clouding of the lung fields on roentgenologic examination of the chest. This is not ordinarily accompanied by signs of pneumonia and has been attributed to pleural edema. The intimate relation of the pleura and fascial spaces, especially the retrovisceral space, is shown in Figure 3. The pleural reaction with edema varies from a minor degree of thickening to a frank pleurisy and the production of sterile, straw-colored fluid. Repeated examinations have never revealed infection of this exudate and it clears up rapidly with subsidence of the mediastinitis.

The use of the procedures described may be followed by prompt healing if all the factors are favorable. However, in spite of diligent care, some unfavorable circumstances may delay recovery and lead to a protracted convalescence. This is well illustrated in Cases 3 and 4, which are presented for contrast.

Case 3.—Hosp. No. 83591: A married woman, age 21, in normal good health, swallowed a fragment of a toothpick which lodged in the cervical esophagus. The foreign body was removed without difficulty through the esophagoscope by Doctor Heatly. The

sharp point, however, was so firmly embedded in the posterior wall that perforation was feared. This was confirmed by the onset of progressive dysphagia, pain, tenderness and emphysema of the neck, fever and leukocytosis of 29,000, during the following 24 hours. Cervical mediastinotomy was performed through an approach in the right side of the neck, and a fulminating *Streptococcus* mediastinitis was drained.

Postoperative treatment was given as described except that oxygen was not needed and the establishment of a gastrostomy was delayed, since it was thought to be unnecessary with such a minute perforation. However, on the fifth postoperative day, the presence of material on the dressing, resembling orange juice, led to a methylene blue test which revealed a fistula. Fluids by mouth were stopped and a gastrostomy was performed. The temperature and pulse reached normal on the ninth day; the drainage diminished and finally ceased on the seventeenth day, and feeding by mouth was resumed. Uninterrupted convalescence followed (Fig. 12B).

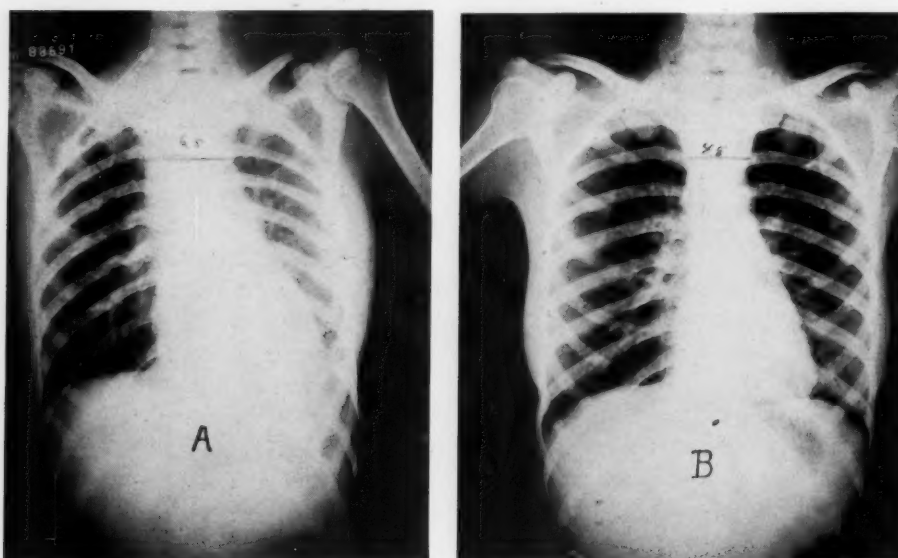


FIG. 12.—(A) Showing the condition, two days after onset, of a suppurative mediastinal infection from esophageal perforation. (B) The appearance of the chest, ten days later, after it had subsided.

This rapid recovery from a virulent infection was no doubt greatly helped by the robust health of this young woman. That the debilitating effect of age and the complication of other disease processes may mitigate against recovery or delay convalescence is well illustrated in Case 4.

Case 4.—Hosp. No. 114,220: A frail woman, age 67, with generalized arteriosclerosis, arteriosclerotic heart disease and cholelithiasis, was admitted for digestive complaints arising from an esophageal diverticulum. Diagnostic esophagoscopy was carried to the opening of the sac but no further. After eating her lunch that day, she developed a chill, fever, leukocytosis, dysphagia, pain, and tenderness; emphysema in the neck soon appeared. A perforation of the esophagus was evident. Operation was performed eight hours after the onset of her first symptom, yet it was found that the retrovisceral space was distended throughout its length with a foul, bloody, purulent fluid containing food particles. This was aspirated out and drains placed to the bottom of the mediastinal cavity. She was placed in an oxygen tent in Trendelenburg position, fluids and blood were given and a gastrostomy established. The temperature, pulse and respiration remained elevated for

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25 days, with persistence of drainage from the wound and fistula in the esophagus. The latter was probably continued by the partial obstruction from the diverticulum. Just after her septic phenomena had returned to normal, they rose again in association with right upper quadrant abdominal pain and jaundice (icteric index 50). A diagnosis of common duct stone was made. Fortunately the stone was passed, and the jaundice gradually cleared up during the following nine days. After draining profusely for 40 days, the mediastinal infection began to subside and cleared completely during the next two weeks (Fig. 13B).

In this patient, the debilitating effect of age with its degenerative disorders and the complications of the cholelithiasis and esophageal diverticulum materially retarded recovery.

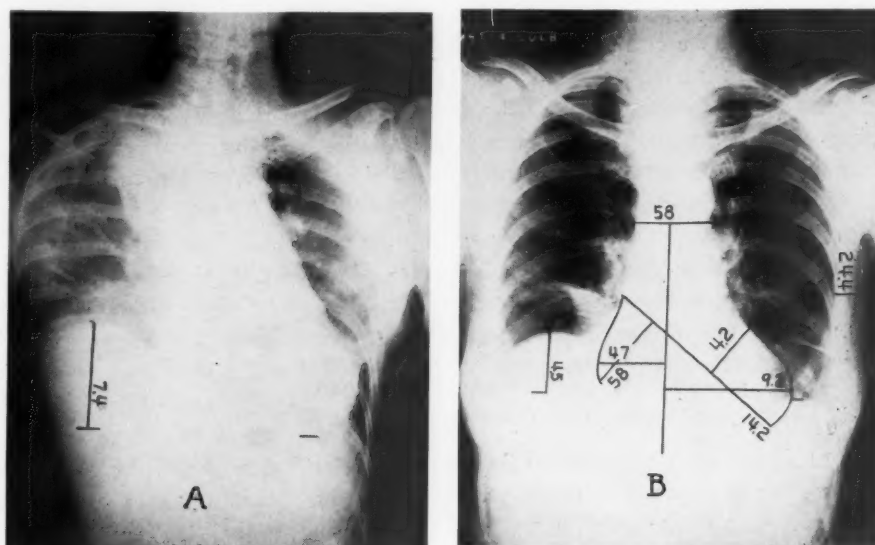


FIG. 13.—Case 4: In (A) an extensive mediastinitis is shown at its worst, while in (B) the process has cleared.

Mediastinitis from cervical sources other than visceral perforation is apt to be slower in its progress and less lethal in its effect. Pus which originates about the mouth or high in the neck, secondary to infection in these regions, tracks down along the fascial spaces slowly enough to give warning. It is often accompanied by an inflammatory reaction in the fascial spaces which produces a barrier to the gravitation of the pus. Care should be used not to break down this barrier at operation, for if it is preserved, recovery will be prompt, as is well illustrated in Case 5.

Case 5.—Hosp. No. 105,448: A robust male, age 17, was admitted with an infection behind the angle of the right mandible, which followed a cold and sore throat two weeks previously. The swelling in the neck was associated with temperature of 40° C., chills, dyspnea, dysphagia and inability to open the mouth. It was drained by the resident surgeon, under the impression that it was a submaxillary space abscess, but in retrospect a parapharyngeal space involvement may have been present. Four days after this operation he had a rise in T.P.R., and complained of tenderness low in the right side of the neck. Operation was advised for descending cervical infection. Thick, yellow pus was

found tracking down mesially to the carotid artery. This showed *Streptococcus hemolyticus* on culture. The cavity extended to the upper part of the mediastinum which was sealed off by inflammatory adhesions. Care was used not to break these down in establishing drainage, and he recovered without further difficulty.

DISCUSSION.—It would appear that suppurative mediastinitis from descending cervical infection is not always a hopeless condition but is amenable to cure if energetic measures are taken to treat it. This requires familiarity with the anatomic arrangement of the cervical fascia, and its spaces, that connect the neck and chest, in order to execute the surgical procedures that are necessary. Persistence in postoperative treatment is most essential.

Anatomic division of the mediastinum by theoretic planes for descriptive purposes is not of much clinical value. Assistance in the solution of practical problems might be gained from dividing the region into four compartments by two planes: the first, a vertical line, would follow the trachea to create the anterior and posterior portions; the second, or horizontal plane, would be at the tracheal bifurcation or about at the level of the sixth dorsal vertebra. This would place the heart and pericardium in the lower anterior quadrant, while the thymus, pretracheal space, substernal thyroid gland, and aberrant parathyroid tumors would be in the upper anterior quadrant. The posterior portion would contain the esophagus and retrovisceral space, the latter divided into its upper and lower portions. From the standpoint of treating suppurative mediastinitis, this concept is useful, for anatomic barriers limit the infection to these four segments.

SUMMARY

(1) Gravitation of pus from the neck causes only one-fifth of the cases of suppurative mediastinitis; in this group, however are found many of the more dangerous infections from visceral perforation.

(2) The paths of dependent spread along the cervical fascial spaces are described. It is found that the suppuration followed the retrovisceral space in 71 per cent, the carotid sheath in 21 per cent, and the pretracheal space in 8 per cent of the cases.

(3) The operative procedures for the prevention and treatment of this type of mediastinitis are discussed. Operation is indicated, as with surgical intervention the mortality is 35 per cent as contrasted with 85 per cent when it is not performed.

(4) Cases of suppurative mediastinitis are presented to illustrate the methods of management of this disorder.

The author's appreciation is expressed to Dr. Clyde A. Heatly, for having made the esophagoscopy examinations, and to Dr. Joseph H. Green, for his roentgenologic work, in the cases reported in this series.

BIBLIOGRAPHY

- ¹ Berry, G.: A Retro-esophageal Abscess from Self-Traumatism. *Laryngoscope*, 34, 120, 1924.
- ² Bichat, X.: *Anatomie Générale*. I. Paris, Brasseur, Gabon and Cie, 1801.

MEDIASTITIS

- ³ Bonn, R.: Zur Operativen Behandlung der Akuten Mediastinal Abszesse. Deutsch. Ztschr. Chir., **158**, 170, 1920.
- ⁴ Butler, E. F.: Putrid Mediastinal Abscess with Spirochetal Infection: Report of a Case. Ann. Oto-Rhino-Laryngol., **43**, 878, 1934.
- ⁵ Cavazzani, G.: Ascesso del Mediastino Posteriore Diagnosticato ed Operato con Toracotomia Dorsale. La Riforma Med., **3**, 110, 1898.
- ⁶ Churchill, Edward D.: Oesophageal Surgery. Surg., Gynec. and Obstet., **60**, 417, 1935.
- ⁷ Coller, F. A., and Yglesias, L.: The Relation of the Spread of Infection to Fascial Planes in the Neck and Thorax. Surgery, **1**, 323, 1937.
- ⁸ Cook, O. S.: Acute Mediastinal Abscess. Am. Jour. Roentgenol., **10**, 696, 1923.
- ⁹ Fisher, R. C.: Abscess of Mediastinum. Jour. Thoracic Surg., **6**, 212, 1936.
- ¹⁰ Furstenberg, A. C.: Acute Mediastinal Suppuration. Tr. Am. Laryng. Rhin. Otol. Soc., **35**, 210, 1929.
- ¹¹ Furstenberg, A. C., and Yglesias, L.: Mediastinitis. Arch. Otolaryngol., **25**, 539, 1937.
- ¹² Gaudiana, V.: The Surgical Treatment of Suppurations in the Posterior Mediastinum. ANNALS OF SURGERY, **63**, 523, 1916.
- ¹³ Glogau, O.: A Typical External Operation for Abscesses Descending from the Upper Air Passages and the Base of the Tongue. Laryngoscope, **32**, 529, 1922.
- ¹⁴ Gutteridge, E. W.: Perforation of the Oesophagus and Mediastinitis. Med. Jour. Australia, **1**, 232, 1931.
- ¹⁵ von Hacker: Zur Operativen Behandlung der Perioesophagealen und Mediastinalen Phlegmone nebst Bemerkungen zur Technik der Collaren und Dorsalen Mediastinotomie. Arch. f. klin. Chir., **64**, 478, 1901.
- ¹⁶ Hare, H. A.: The Pathology, Clinical History and Diagnosis of Affections of the Mediastinum. Philadelphia, 1889.
- ¹⁷ Hayes, H. M.: Cellulitis of Pharyngeal Tissues; Mediastinitis; Death. Laryngoscope, **42**, 128, 1932.
- ¹⁸ Heatly, C. A., and Pearse, H. E.: The Management of Perforations of the Cervical Esophagus. Ann. Otol. Rhin. and Laryngol., **41**, 1235, 1932.
- ¹⁹ Heatly, C. A.: Minute Perforation of the Cervical Esophagus; Fulminating Descending Infection; Mediastinitis; External Operation; Recovery. Ann. Otol. Rhin. and Laryngol., **43**, 873, 1934.
- ²⁰ Heidenhain, L.: Über einen Fall von Mediastinitis Suppurative Pastica nebst Bemerkungen ueber die Wege, ins hintere Mediastinum einzudringen. Arch. f. klin. Chir., **59**, 199, 1899.
- ²¹ Henke, P. J. W.: Beitrage zur Anatomie des Menschen mit Beziehung auf Bewegung. Leipzig, C. F. Winter, **1**, 12, 1872.
- ²² Iglaue, S., and Ransohoff, J. L.: Perforation of the Esophagus by a Foreign Body, with Report of a Case Presenting Unusual X-Ray Signs. Laryngoscope, **34**, 821, 1924.
- ²³ Iglaue, S.: Surgical Approaches to Deep Suppuration in the Neck and Posterior Mediastinum. Arch. Otol., **21**, 707, 1935.
- ²⁴ Jones, C. C., Brown, S., and Fine, A.: Mediastinal Abscess Complicating a Retropharyngeal Abscess. Radiology, **28**, 747, 1937.
- ²⁵ King, E.: Perforation of the Esophagus with Report of Six Cases. Ann. Otol. Rhin. and Laryngol., **38**, 351, 1929.
- ²⁶ Knaggs, R. L.: Perforation of the Esophagus by a Rabbit Bone. Lancet, **1**, 933, 1908.
- ²⁷ König, F., and Riedel, B.: Die Entzündlichen Processe am Hals und die Gerschwulst am Hals. Deutsch. Chir., **36**, 1882.
- ²⁸ Konjelzny, G. E.: Ein Beitrag zur Kenntnis und Chirurgischen Behandlung der Phlegmonosen Mediastinitis. Deut. Zeitschr. Chir., **192**, 108, 1926.
- ²⁹ Kornblum, K., and Osmond, L. H.: Mediastinitis. Amer. Jour. Roentgenol., **32**, 23, 1934.

- ⁸⁰ Kramer, H.: Statistisches zur Frage der Heilbarkeit des Mediastinal Abszesses. *Ztsch. f. Laryngol.*, **25**, 333, 1934.
- ⁸¹ Lambert, A. V. S., and Berry, F. B.: The Mediastinum. *Arch. Surg.*, **14**, 261, 1927.
- ⁸² Lederer, F. L., and Fishman, L. Z.: Prophylactic Mediastinotomy for Perforating Esophageal Foreign Bodies. *Arch. Oto.-Laryngol.*, **19**, 426, 1934.
- ⁸³ Lerche, W.: Surgical Treatment of Suppuration in Posterior Mediastinum. *Surg., Gynec. and Obstet.*, **32**, 232, 1921.
- ⁸⁴ Lerche, W.: Suppuration in the Posterior Mediastinum. *Arch. Surg.*, **8**, 247, 1924.
- ⁸⁵ Lerche, W.: Infected Mediastinal Lymph Nodes as a Source of Mediastinitis. *Arch. Surg.*, **14**, 285, 1927.
- ⁸⁶ Lilienthal, H.: Posterior Mediastinotomy. *Arch. Surg.*, **6**, 274, 1923.
- ⁸⁷ Lurman: Ein Fall von Oesophagusfistel mit Secundärer Bildung eines Mediastinal Abszesses. *Berlin klin. Wchnschr.*, **13**, 257, 1876.
- ⁸⁸ Mainzer, F. S.: Mediastinitis Following Removal of Thyroid. *Am. Jour. Surg.*, **8**, 817, 1930.
- ⁸⁹ Marschik, H., and Voge, H.: Fremdkörper in dem Oberen Luft und Speisewegen mit Besonderer Berücksichtigung der Gesaphagotomie. *Wien. klin. Wchnschr.*, **22**, 1405, 1909.
- ⁹⁰ McGinnis, E.: Mediastinitis as an Occasional Resultant Complication of Foreign Bodies in the Esophagus. *Laryngoscope*, **34**, 831, 1924.
- ⁹¹ Meyer, P.: Erfolgreiche Kollare Mediastinotomie bei Akutem Traumatiscnem Mediastinalempysem und bei Mediastinal Phlegmone. *Schweizer Med. Wchnschr.*, **63**, 931, 1933.
- ⁹² Meyersburg, H.: Traumatic Perforation of Esophagus with Mediastinitis. *J.A.M.A.*, **95**, 1341, 1930.
- ⁹³ Moersch, H. J., and Kennedy, F. S.: Mediastinitis. *Med. Clin. North Amer.*, **16**, 1433, 1933.
- ⁹⁴ Mosher, H. P.: The Submaxillary Fossa Approach to Deep Pus in the Neck. *Tr. Am. Acad. Ophthalmol.*, **34**, 19, 1929.
- ⁹⁵ Müller, W.: Mediastinalabszesse. *Zentralbl. f. Chir.*, **47**, 231, 1921.
- ⁹⁶ Myerson, M. C.: Perforations of the Esophagus. *Ann. Otol. Rhin. and Laryngol.*, **37**, 545, 1928.
- ⁹⁷ Neuhof, H.: Acute Infections of the Mediastinum with Special Reference to Mediastinal Suppuration. *J. Thoracic Surg.*, **6**, 184, 1936.
- ⁹⁸ Obalinski, A.: Beitrag zur Operativen Behandlung des Hinteren Brustfellraumes. *Wien. klin. Wchnschr.*, **9**, 1175, 1896.
- ⁹⁹ Orton, H. B.: Mediastinitis Following Esophageal Foreign Body. *Arch. Otolaryng.*, **12**, 635, 1930.
- ¹⁰⁰ Palmer, F. E.: Descending Abscess in the Neck. *J.A.M.A.*, **83**, 2067, 1924.
- ¹⁰¹ Parsons, F. G.: On the Carotid Sheath and Other Fascial Planes. *J. Anat. and Physiol.*, **44**, 153, 1910.
- ¹⁰² Pearse, Herman, E., Jr.: The Operation for Perforation of the Cervical Esophagus. *Surg., Gynec. and Obstet.*, **56**, 192, 1933.
- ¹⁰³ Pearse, Herman E., Jr.: The Management of Infections of the Neck and Their Complication: Mediastinitis. *Jour. Missouri State Med. Assn.*, **35**, 69, 1938.
- ¹⁰⁴ Poulsen, K.: Über die Fascien und die Interfascialen Räume des Halses. *Ztschr. f. Chir.*, **23**, 223, 1886.
- ¹⁰⁵ Poulsen, K.: Über Abscesse am Halse. *Ztschr. f. Chir.*, **37**, 55, 1893.
- ¹⁰⁶ Rasumowski: Ein Fall von Mediastinitis Acuta Purulenta Postica in Folge von Schussverletzung des Oesophagus mit Ausgang in Genesung. *Ljetopis russkoi chir.*, **6** (abstracted in Jahresbericht f. Chir., **5**, 412, 1899).
- ¹⁰⁷ Richards, L.: Peritracheal Abscess. *Arch. Otolaryngol.*, **11**, 336, 1930.
- ¹⁰⁸ Sautter, C. M.: Ludwig's Angina and Mediastinal Abscess Following Tonsillectomy. *J.A.M.A.*, **87**, 1831, 1926.

- ⁵⁹ Schinz, H. R.: Über einen Senkungs abszess im Vorderen Mediastinum. Deutsch. Ztschr. Chir., **159**, 163, 1920.
- ⁶⁰ Schmitt, Adolph: Die Fascienseiden und ihre Beziehungen zu Senkungs Abscessen. Munchen, J. F. Lehmann, 1893.
- ⁶¹ Soltman, O.: Die Ausbreitungsbezirke der Congestionsabscesse bei der Spondylarthrocace der Kinder. Jahrb. f. Kinderh., **7**, 267, 1874.
- ⁶² Spiess, G.: Forderung nach Frühzeitiger Röntgendurchleuchtung mit Kontrastmitteln bei Verschluckten Fremkörpern. Ztschr. f. Hals-Nasen-Ohren, **21**, 527, 1928.
- ⁶³ Treitel: Über das Wesen und die Bedeutung Chronischer Tonsillarabscesse. Deutsch. med. Wchnschr., **24**, 761, 1898.
- ⁶⁴ Vogel, K.: Durch Endoskopische Oesophagus-Spaltung Geheilte Mediastinaler Senkungs-Abszess. Ztschr. f. Laryngol., **22**, 317, 1932.
- ⁶⁵ Wagner, L. C.: Posterior Mediastinal Abscess Following Suppurative Arthritis of Cervical Vertebrae. ANNALS OF SURGERY, **87**, 511, 1928.
- ⁶⁶ Wietke, C.: Über Oesophagus Perforationen Nervorgerufen durch Oesophagaskopie. Ztschr. f. Hals-Nasen-Ohren, **28**, 58, 1930.
- ⁶⁷ Ziembicki, G.: Du Phlegmon du Mediastin Postérieur et de son Traitement. Bull. et Mém. Soc. Chir., **21**, 190, 1895.

DISCUSSION.—DR. EDWARD D. CHURCHILL (Boston, Mass.): Perforation of the esophagus by foreign body, or as a result of instrumentation, is as urgent a surgical emergency as a perforated abdominal viscus. The flora of the lumen of the esophagus is as virulent as that of the large bowel. The proper treatment of perforation of the cervical and upper thoracic esophagus is immediate operation by the cervical approach. Operation should not be delayed for the development of signs of local inflammation or roentgenologic evidence of air in the tissues.

Unfortunately, perforation of the esophagus frequently occurs as one of the tragedies of modern medicine—a dangerous and possibly fatal complication of a diagnostic procedure. A perforation of the esophagus by instrumentation must be handled like a perforation of any other viscus. If a surgeon is so unfortunate as to perforate the sigmoid with the endoscope, he knows what he must face and what he must do. Endoscopists must realize that if they find a perforation from a foreign body or if they are unfortunate enough actually to perforate the esophagus with their instrument, proper treatment must be carried out immediately.

I have used the method employed by Doctor Pearse, in an elderly patient with a chicken bone perforation, demonstrated by esophagoscopy. She was diabetic, and operation was performed within 15 hours of the perforation. There was already foul-smelling, thin pus in the retrovisceral space. Recovery followed.

The virtues of posterior mediastinotomy in approaching abscesses of the retrovisceral space must not be overlooked, and while the cervical approach, as Doctor Pearse has shown, is the method of election in perforations of the cervical esophagus and perhaps the upper mediastinal portion of the esophagus, there will be an occasional case where posterior mediastinotomy should be employed. I have performed this twice, and one of the two patients recovered.

A problem that Doctor Pearse did not touch upon, but concerning which we would like information, is the handling of perforations of the esophagus that occur below the level of the bifurcation of the trachea. After infection occurs, the treatment is obvious, but should immediate mediastinotomy be advised if the case is seen early? Usually such a case is seen only after an empyema has developed secondary to a mediastinitis.

Doctor Pearse brings out the problem of feeding, and states that he per-

forms a gastrostomy in many instances. Personally, I have employed a gastric tube which is inserted on the operating table, while a finger can be kept in the incision, and the tube guided past the point of perforation.

There is one other quite rare route for extension of cervical infection into the thorax. Suppuration arising from cervical lymphangitis in the region of the juncture of the great veins just at the thoracic inlet may, instead of descending medially into the mediastina, descend laterally, dissecting the parietal pleura from the endothoracic fascia and present in the axilla, simulating an empyema.

DR. MARTIN B. TINKER (Ithaca, N. Y.): I would like to compliment Doctor Pearse on bringing before this Association this very dangerous and rather difficult complication which is likely to occur to any of us, particularly those who do much surgery in the neck.

A condition which has come to my notice was not mentioned: Extension down the jugular vein of infection in thrombosis of the lateral sinus. Another case had a perforation of the esophagus by an oyster shell, which caused extensive infection; and two cases of neglected suppurative thyroiditis resulted in a serious descending infection. It is unbelievable how rapidly these infections develop in some instances.

DR. HOWARD LILIENTHAL (New York, N. Y.): This is a matter which, as a thoracic surgeon, has interested me tremendously. I have written a little about it and you will find some points which have been brought out by this excellent paper of Doctor Pearse's in a book that I¹ wrote, which appeared 13 years ago.

Although I have discussed this repeatedly since that time, it appears to me that surgeons in general are afraid of the mediastinum. I believe that if there is a history which points to the possibility of this complication, it is better not to wait, particularly when the patient is very ill. Posterior mediastinotomy is not nearly as dangerous as many seem to believe. If no infection happens to be encountered, there is, with proper precautions, almost no likelihood that it will become infected from the exploration. Posterior mediastinotomy affords an excellent exposure.

I would emphasize one or two points in regard to this form of exploration: (1) If there is a history of endoscopy, especially of the esophagus, one should early suspect involvement of the mediastinum, and one should not fear mediastinotomy by the posterior approach. If the suspected lesion is in the lower part of the esophagus, exploration is just as necessary as when it is in the part with which Doctor Pearse's paper especially deals.

Gunshot wounds may also infect the mediastinum, posterior or anterior, and even operations for goiter have been known to be followed by this complication; although I myself have seen only one. I agree with the speaker about the importance of inverting the patient, but in some instances where this is difficult, a multifenestrated tube placed through the upper wound into the space and equipped with a suction apparatus will be effective. This will often make a secondary procedure unnecessary. It should also be borne in mind that in the lower third of the chest the right pleura often, in fact usually, extends over the midline into the left. Therefore, in opening the lower part of the mediastinum, especially on the left side, beware of the overlapping right pleura, or there may be a fatal infection of both cavities.

Another useful function, if one may so denominate it, of posterior mediastinotomy is the opening of certain lung abscesses which point here. I have done this several times.

Finally, I would urge that the possibility of suppurative mediastinitis should be borne in mind, even though roentgenologic examination does not reveal it.

REFERENCE

- ¹ Lilienthal, Howard: Thoracic Surgery, Saunders, 1925.

DR. JOHN ALEXANDER (Ann Arbor, Mich.): May I offer, as a corollary to Doctor Pearse's presentation, a suggestion as to the choice between cervical drainage and thoracic drainage of these abscesses? I have used both approaches and have been satisfied with both, but I feel one should choose the approach according to the case. May I suggest that in those which are operated upon quite early after the beginning of the infection, and are located high in the mediastinum or low in the neck, the cervical approach is obviously the better; but those that have localized as far down as the fifth or sixth thoracic spine, particularly when the case is a relatively old one, let us say more than two or three weeks old, and particularly when there is evidence that some foreign body has lodged in the abscess, a posterior mediastinotomy is the better approach.

I feel that in a case that has become subacute or chronic, prolonged cervical drainage through a tube which lies close to the carotid sheath may possibly cause an erosion of the carotid artery and fatal hemorrhage, whereas, prolonged drainage through a wide posterior mediastinal incision, with packing of that incision, presents almost no danger of pressure necrosis of a large vessel.

As Doctor Lilienthal has said, the opening of the mediastinum is simple in cases in which there is a large abscess that projects on either side of the spine, or bilaterally. The parietal pleura will have been dissected away from the mediastinum by the abscess, so that a resection of two or three inches of two posterior ribs together with the transverse vertebral processes, and a division of the intervening intercostal bundle, permit one to enter the abscess directly. If a foreign body has dropped into the bottom of such an abscess, or is imbedded in the soft tissues, it would be difficult to safely remove it if one attempted to do so through the more or less long track which would have been produced through a cervical incision.

In summarizing, I suggest the use of a cervical incision in acute cases and in those in which the infection, particularly free pus, has not extended as far as the fifth or sixth thoracic spine, but a posterior thoracic incision for older cases which will probably require prolonged drainage and those in which a foreign body may have become lodged in the abscess in a position that is relatively inaccessible through a cervical incision.

DR. OWEN H. WANGENSTEEN (Minneapolis, Minn.): This presentation by Doctor Pearse is an important one. There is one point which I should like to make concerning early recognition of perforation of the esophagus. Doctors Pearse and Churchill both indicated that there was often much delay between the occurrence of this tragedy and its identification. Whereas, considerable time may elapse before subcutaneous emphysema becomes appreciable by palpation, yet, the presence of air in the interstitial tissues about the esophagus can usually be detected in a roentgenogram a few minutes after its escape from the esophagus. I would, therefore, strongly urge that all cases in which perforation of the esophagus is suspicioned have a roentgenogram made immediately of the neck and thorax in order to identify the possible presence of air. Such a film can be repeated if necessary after a short elapse of time.

A hospital visitor with a self-inflicted bullet wound through the neck, who had shot a hospital patient through the chest, was seen by me several years ago, a short time after the shooting. The offender presented no evidence of hemorrhage or nerve injury and there was no subcutaneous emphysema. He was hustled off to jail and died a few days later, I learned subsequently, from mediastinitis resulting from perforation of the esophagus. Since then, I have looked for evidence of escaped air from the esophagus in a roentgenogram rather than with the palpating hand. In the drowsy small hours of the night, I have once made the diagnosis of spontaneous perforation of the lower esophagus over the telephone in conversation with the surgical resident, on the basis of subcutaneous emphysema in a patient who had upper abdominal pain, no dyspnea or cough. The roentgenogram, as I have indicated, is, however, a much earlier and a better determinant of the presence of air in the para-esophageal interstitial tissues.

I would like to cite an unfortunate happening attending operative closure of a cervical perforation of the esophagus which occurred after esophagoscopy, in the interest of those who may be disposed to try something of the sort. At the time of operation, the thought occurred to me that if the carotid sheath were opened and the smooth inner surface of that sheath sutured over the site of suture of the perforation, the risk of mediastinitis would be lessened. The patient was maintained in the Trendelenburg posture and no mediastinal abscess developed. The patient's convalescence seemed assured, and somewhat more than a week later, I withdrew a soft rubber tissue drain (Penrose) which had been left in the wound. A slight blood stain was noted on the dressing subsequently, but a few hours later the patient died suddenly from profuse bleeding from the wound. I had anticipated finding the source of hemorrhage in an intervertebral vein or from the left internal jugular at postmortem. Much to my astonishment and amazement, however, the erosion was in the common carotid artery. Whereas, the use of the carotid sheath may secure the esophageal suture line, it is decidedly not a safe procedure.

This experience leads me to make a brief digression. It seems a bit odd that the thick walled artery should have ruptured rather than one of the adjacent thin walled veins. It seems to me that this very occurrence sheds some light upon the shortness of our years. Our arteries are called upon to sustain a relatively great pressure unrelentingly over years. Is it not likely that arteriosclerosis may be largely a traumatic process? If it is metabolic in origin, who do not veins exhibit these age changes in the same measure as it is observed in arteries? It may well be that the biblical three score and ten years are determined by the length of time that arteries will withstand the effects of systolic blood pressure.

I have had a single experience with an esophageal fistula resulting from pressure by a mediastinal abscess. What astounded me most about it was the great difficulty in securing closure of the fistula. After the performance of gastrostomy, I waited months for it to close spontaneously; no esophageal obstruction could be demonstrated. Yet this fistula stayed open until a very extensive decostaliation, performed in stages, closed eventually both a total empyema and the esophageal fistula.

DR. HERMAN E. PEARSE (closing): Doctor Churchill has brought up the matter of after-care, particularly in relation to the feeding of patients with esophageal lesions. This is very important, for fluid balance must be maintained; blood is often needed, not only to supply hemoglobin but also deficient serum protein and oxygen may be required for prolonged periods in a tent.

MEDIASTINITIS

Feeding may be difficult. I have had a discouraging experience with the duodenal tube, largely because the patient is already uncomfortable from the wound, the manipulation of the oxygen tent, and the administration of fluids. I have tried it repeatedly and have now returned to a gastrostomy as the simplest procedure.

I would agree entirely with Doctors Churchill's and Alexander's presentation that these patients must be individualized, and if I may be allowed to predict, I believe that with more experience, we will divide them into three groups: One will constitute those infections which slowly gravitate down fascial planes from the neck. They should be simply opened and drained. Nature's barrier against the spread of infection should not be broken down. In the next group are those who have either a spreading infection or a localized abscess below the level of the fifth or sixth dorsal vertebra. They certainly should be attacked through the chest wall. The fascial spaces from the neck extend only to the level of the sixth vertebra, so one cannot use the cervical incision for those at the lower level. The same approach should be used for long-standing abscesses in any location, for they need some collapsing of the chest wall in order to obliterate the cavity.

The third group are those described who have a virulent spreading supuration from a visceral perforation. Their treatment must be much more energetic than for a localized abscess, and if the perforation is above the sixth dorsal vertebra, the mediastinum should be drained immediately through the neck.

I have never encountered the complication of hemorrhage spoken of by Doctors Alexander and Wangenstein. We all know that Doctor Halsted devised a very ingenious method for the gradual occlusion of great vessels with an aluminum band. He stated that he had cured aneurysms but some patients died of hemorrhage; therefore, it has long been known that gradual pressure on a pulsating vessel will wear away its wall.

Two factors might increase the risk of hemorrhage: One is the dissection of the carotid artery to expose its wall in the incision. This is not necessary. It should be left alone with its fascial envelope intact. The second is the use of a hard drainage tube. In any experience I have had in operative vascular surgery, I have never seen soft material erode a vessel wall.

THE MANAGEMENT OF CERTAIN LESIONS OF THE ESOPHAGUS

GROVER C. PENBERTHY, M.D., AND CLIFFORD D. BENSON, M.D.

DETROIT, MICH.

FROM THE SURGICAL SERVICES OF HARPER HOSPITAL AND THE CHILDREN'S HOSPITAL OF MICHIGAN, DETROIT, MICH.

LESIONS of the esophagus resulting from foreign bodies, diverticula, lye burns, spontaneous rupture and ruptures resulting from direct violence are not common, when one considers how few cases of this type are seen when compared with other surgical lesions of the gastro-intestinal tract. A large percentage of the cases classified in this group are not seen by the surgeon early in the life of the lesion or injury, and when the surgeon is called, it is often after some complication has developed.

Today, the management of these lesions is well standardized after the contributions made by Jackson,² Judd,⁸ Lahey,⁴ Pearse,³ Shallow,⁷ Truesdale¹ and others. There may be some difference of opinion as regards the one or two stage operation, where esophagotomy is necessary. The two stage operation advocated by Judd and Lahey is generally considered the safest procedure and has been practiced in the cases to be presented in this paper.

The resultant danger from perforation of the esophagus, regardless of the location, becomes a serious complication. Rupture in the thoracic portion develops contamination of the mediastinal structures and a virulent infection, with few patients able to survive, in spite of surgical drainage. The mediastinitis following cervical perforation as described by Pearse is an indirect effect. The explanation of Pearse and others is that it results from a dependent spread of infection from the neck into the chest along the fascial spaces. There is abundant evidence to prove that a direct communication exists between the cervical region and the mediastinum. This is due to the fact that, during embryologic development, the mediastinal structures originate in the neck and migrate into the chest, carrying their enveloping fascia with them. The conception that the mediastinum begins at the diaphragm and ends at the base of the skull is logical since at no place is there a transverse demarcation to segregate these regions. This anatomic relationship emphasizes the need for protection against a spread of infection from the cervical region to the mediastinum, when dealing with lesions involving the cervical region of the esophagus requiring esophagotomy.

The addition of esophagoscopy, as practiced by the skilled endoscopist, has contributed to the diagnosis and management of this type of case. The close cooperation of the endoscopist, roentgenologist and surgeon is essential in the successful appreciation of the problems involved and the ultimate result.

A foreign body lodging in the esophagus, or a lesion resulting from a foreign body, is the one most frequently seen by the endoscopist. The refinement of technic as practiced today fortunately minimizes the need for open operation. The work of Chevalier Jackson and his associates stands

out preeminently in the development of an excellent operative technic, which has resulted in a surprisingly low mortality of 2 per cent or less. He regards the esophagoscope, in the hands of rough, careless, unskilled physicians, as a dangerous and often fatal instrument. Moreover, there are risks associated with the use of the esophagoscope which he describes as "complications and dangers." Jackson has stated that "endoscopic skill cannot be bought with instruments. Repeated exercise of a particular series of maneuvers is necessary. As with learning to play a musical instrument, a fundamental knowledge of technic, positions, and landmarks is necessary, after which only continued manual practice makes for proficiency."

The management of pulsion esophageal diverticula, as described by Lahey, shows a refinement in technic, with gratifying results as shown in his follow-up of the cases operated upon. He has emphasized the need for early recognition of this lesion and surgical treatment, preferring the two stage operation, at the same time recognizing the results obtained by the advocates of the one stage operative procedure.

The great majority of cicatricial stenoses of the esophagus are seen in children as a result of the accidental swallowing of lye. When these cases are first seen, they should be treated conservatively, allowing only bland fluids for a period of about two weeks. The resultant strictures of the esophagus are often multiple, and may be resistant to treatment. Early dilatation of strictures of the esophagus has been practiced by Salzer and Bokay with satisfactory results. Most observers prefer waiting until the acute inflammatory reaction has subsided.

Where no gastrostomy is performed, we believe Jackson's peroral esophagoscopic bouginage for cicatricial stenosis, under direct vision, is safe and produces good results. Blind bouginage is to be strongly condemned. Where dilatation from above is impossible, the surgeon is called upon to perform a gastrostomy, not only for feeding purposes, but for future retrograde dilatation. Tucker claims this the safest method, and the results are more permanent and more rapidly attained. The Stamm type of gastrostomy has been found satisfactory, precaution being taken to select the site near the cardia, midway between the greater and lesser curvatures of the stomach.

The following case reports illustrate some of the problems encountered and the management of certain surgical lesions of the esophagus.

FOREIGN BODIES

Case 1.—J. C., white, male, age 3, was admitted to the hospital March 2, 1936, with a history of having swallowed an alarm clock key that day. The child soon coughed and choked. He was referred to the endoscopist and later fluoroscoped, the key being found at the level of the fifth cervical vertebra (Fig. 1).

Esophagoscopy was carried out by Dr. H. Lee Simpson, who visualized the foreign body below the level of the cricoid. It was so tightly incarcerated and fixed that it could not be turned; removal by this route, therefore, was considered impossible. The following day, March 3, 1936, a first stage external esophogotomy was performed.

Operation.—The esophagus was exposed, two long black silk sutures were inserted into the wall for subsequent identification, and the wound packed. The patient was given

intravenous glucose and saline subcutaneously for the next few days. On March 8, 1936, the second stage esophogotomy was performed under general anesthesia. The foreign body could not be felt, but a roentgenogram showed it to be at the level of the fourth cervical vertebra. A one-half inch incision was made in the esophagus, and with the aid of a Cameron light, the foreign body was seen, grasped with a forceps, and gently removed, under vision. The defect in the esophagus was closed by interrupted catgut sutures. The wound was closed in layers, with a small rubber drain inserted at the lower angle. A Levine tube was passed for feeding purposes and removed 11 days later.

Subsequent Course.—There was no noticeable drainage from the wound. The patient was discharged 20 days postoperatively. One month later, he was reported to be in good condition.



FIG. 1.—Roentgenogram showing an alarm clock key in the esophagus at the level of the fifth cervical vertebra.

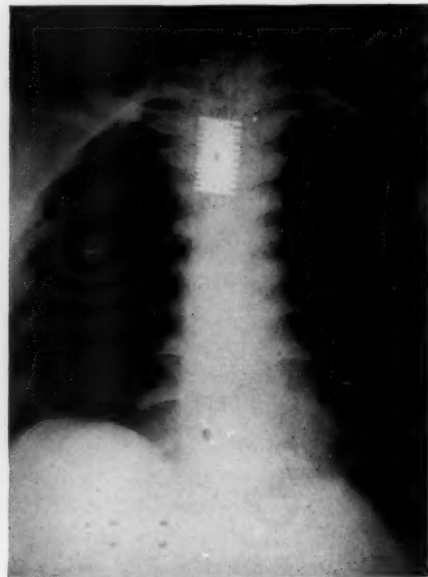


FIG. 2.—Roentgenogram showing a razor guard in the esophagus at the level of the transverse arch of the aorta.

Case 2.—T. D., white, male, age 46, was admitted to the hospital December 12, 1934, with a history of having been in an institution during the previous five months because of a mental condition. One month prior to admission to Harper Hospital, he had attempted suicide by swallowing the two parts of a safety razor which hold the blade. One piece was reported as having been passed per rectum. Following the swallowing of the safety razor guard, he had intermittent attacks of severe substernal pain. Fluoroscopic examination revealed a metallic guard from a safety razor in the lower esophagus, about four inches above the cardiac end. Temperature, 102° F.; pulse, 100; respirations, 24. *Physical Examination* did not reveal any evidence of cyanosis, or emphysema of the soft tissue of the neck and thorax. The following day, he was esophagoscoped by Dr. A. E. Hammond. A 9 Mm. esophagoscope was passed, and about three inches above the diaphragm, the esophagus was observed to be studded with granulation tissue, which was partially removed, and the metal foreign body visualized. This was grasped with a heavy, forward-grasping forceps, and gently pulled upward. It was brought to the level of the cricoid cartilage, at which point it was impossible to pull it further. The following day, roentgenologic examination of the chest, in the anteroposterior and lateral planes, revealed the safety razor guard lying at the level of the transverse arch of the

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aorta (Fig. 2). At this time, there was no evidence of emphysema, and both lung fields were normally aerated.

A roentgenologic examination of the chest four days later showed the guard to have descended about two inches since the former examination. The patient was again esophagoscoped, December 19, 1934, and a 9 Mm. esophagoscope was passed. With the Toddle forceps, the operator was able to grasp the foreign body and it was elevated to the level of the cricoid cartilage, but again it was found impossible to extricate it further. On December 23, 1934, it was decided that the foreign body should be removed by the external route.

Operation.—December 26, 1934: The first stage operation was performed, which simply exposed the esophagus. A gauze pack was inserted down to the esophagus, and the wound closed. Convalescence was uneventful; and five days later the second stage operation was performed. The esophagus was exposed, and a one-half inch incision was made through its lateral aspect. A short esophagoscope was then introduced, orally, and the foreign body was elevated to the level of the surgical incision in the esophagus, through which it was extracted. The wound in the esophagus was closed, and a small soft rubber drain inserted. A Levine tube was passed for feeding purposes. Convalescence was uneventful, except for a small amount of drainage from the operative wound; and there was no evidence of an esophageal fistula. The Levine tube was removed at the end of 12 days. The surgical incision was completely healed January 16, 1935, at which time the patient was discharged from the hospital.

Follow-Up.—The patient has been seen on a number of occasions and has returned to his former occupation. A fluoroscopic and roentgenologic examination with a barium meal, five weeks after operation, showed the barium to have passed through the esophagus without any evidence of obstruction, but there was present a moderate amount of spasm (Fig. 3). There is no difficulty in swallowing; no evidence of stricture; and he is apparently normal mentally.

Case 3.—A. P., white, female, age 44, married, entered the hospital April 21, 1937, with a history of having swallowed a fish bone a few hours previously. Roentgenologic examination revealed the presence of a small bone in the esophagus at the level of the sixth and seventh cervical vertebrae. It was also noted that there was some increased radiotransparency about the bone, suggesting the probability of some air in the peri-esophageal tissues. The patient was esophagoscoped by Dr. Arthur E. Hammond, who visualized the fish bone lying cross-wise in the cervical region of the esophagus. It was impossible to extract it with fine, grasping-forceps. The patient was, therefore, returned to the ward, and fluids administered by both intravenous and subcutaneous routes. Two days later, the patient's general condition appeared critical: Temperature, 101° F.; pulse, 108; respirations, 22. There was also noted a moderate cyanosis of the face, upper thorax and neck; pitting edema in both upper and lower extremities; and clinical evidence of emphysema in both cervical regions, which extended to the level of the clavicle on the left side. Because of the cyanosis and dyspnea, the patient was placed in an oxygen tent, and treatment instituted to restore her fluid balance in preparation for the



FIG. 3.—Roentgenogram taken six weeks after the removal of the foreign body by the external route. There is no evidence of stricture but a moderate degree of spasm.

first stage external esophagotomy. A periesophageal abscess was suspected as being secondary to a perforation of the cervical esophagus.

Operation.—April 24, 1937, three days after admission: A left, first stage external esophagotomy was performed under nitrous oxide anesthesia. Upon exposing the esophagus at the level of the foreign body, there was found a walled-off abscess at the postero-lateral aspect of the esophagus (Fig. 4A).



FIG. 4.—(A) Drawing of the periesophageal abscess found at operation. (B) The small perforation in the wall of the esophagus which was seen after drainage of the periesophageal abscess.

The wound was packed anteriorly, inferiorly and posteriorly, and the abscess drained. The culture of the pus was reported as *Staphylococcus aureus*. At this operation, a small perforation in the wall of the esophagus was seen (Fig. 4B). A small rubber drain was inserted to the site of the perforation and placed in the lower angle of the wound. The patient's postoperative condition was satisfactory, and the fluid requirements were met by glucose intravenously and saline subcutaneously. There was a small amount of drainage of a serosanguineous type from the wound during the next few days. Roentgenologic examination, April 28, 1937, revealed no definite evidence of the fish bone shadow in the cervical region. At this time the patient was esophagoscoped by Doctor Hammond, who reported the cervical portion of the esophagus to be slightly inflamed and after careful inspection, found no tear in the esophageal wall, or foreign body. The

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esophagoscope was passed gently down to the cardia. It was concluded that the foreign body had passed down into the gastro-intestinal tract. A Levine tube was passed for feeding purposes, and was removed May 4, 1937, when fluids were given by mouth. There was apparent need for maintaining drainage for 11 days, at the end of which time the temperature, pulse and respirations were normal, and the patient was discharged. Recovery was uneventful. At the present time, the patient is reported in good health and has had no symptoms referable to the esophagus.

ESOPHAGEAL DIVERTICULUM

Case Report.—H. A., white, male, age 60, first came under observation October 19, 1932, with a history of having had difficulty in swallowing for two years. This difficulty had been progressive in type, and was not associated with any hoarseness. He stated that a fulness in the lower left neck region appeared usually after his evening meal. Three or four hours later, by exerting pressure over this area of fulness, he was able to express previously eaten food into the mouth. At this time, the patient was not acutely ill and weighed 121 pounds. Examination showed a slight fulness in the neck in the region of the left lobe of the thyroid. He was referred for roentgenologic study October 24, 1932. Fluoroscopic examination demonstrated a very definite distention of the upper third of the esophagus, with a large diverticulum bulging from the posterior aspect of the esophagus just at the suprasternal notch (Fig. 5). The barium trickled through the esophagus below this point, and there was no indication of any other abnormality.

Operation.—October 28, 1932: A first stage external esophagotomy was performed under bilateral paravertebro-cervical nerve block anesthesia. The left lobe of the thyroid was mobilized and retracted to the midline, exposing the diverticulum. The latter was freed along its entire length down to its communication with the esophagus. The sac was anchored to the prethyroid muscle, iodoform gauze was packed around the diverticulum and was brought out through the lower end of the wound. A Levine tube was passed for feeding purposes. Convalescence, after the first stage operation, was uneventful, except for a small amount of seromucous drainage from the wound. There was apparently a small tear made in the wall of the diverticulum at the time of its isolation, resulting in a small esophageal fistula. On November 16, 1932, 18 days later, the second stage operation was performed. The sac was excised, and the small defect in the esophagus was closed. The patient was fed by a Levine tube until November 20, 1932, when the tube was removed and fluids allowed by mouth. The drains were removed from the wound November 21, 1932, and the patient was discharged. When last seen, in the spring of 1937, the patient reported no difficulty in swallowing, and was in good general health except for a mild hypertension.

CONGENITAL SHORTENING OF THE ESOPHAGUS

Case Report.—L. S., male, age seven weeks, was admitted March 29, 1933, with a history of vomiting after taking his formula from the time he was one week old. The vomiting occurred one-half to two hours after the feedings, and occasionally was projectile in type. The vomiting continued and fluoroscopic and roentgenologic studies were made April 3, 1933, and April 23, 1933, which demonstrated a congenitally short esophagus and a pouch-like projection of the stomach above the diaphragm (Fig. 6).

From the clinical and roentgenologic findings, it was decided to perform a left phrenic crushing procedure, which was effected April 29, 1933. All vomiting ceased thereafter, and the child has had no further difficulty.

STRICTURE OF THE ESOPHAGUS

Case Report.—L. G., colored, female, age 3, was admitted to the Children's Hospital June 12, 1935, with a history of having swallowed lye about ten weeks previously. Since then, she had considerable difficulty in swallowing. A diagnosis was made of an esophageal stricture secondary to a lye burn. Roentgenologic examination demonstrated the presence of a complete stricture of the lower third of the esophagus (Fig. 7).



FIG. 5.—Roentgenogram showing an esophageal diverticulum measuring 4 cm. in diameter.



FIG. 6.—Roentgenogram showing the short esophagus and a portion of the stomach lying above the diaphragm. There is also to be noted a distinct dilatation of the esophagus and a pouch-like projection from both aspects of the stomach above the diaphragm.

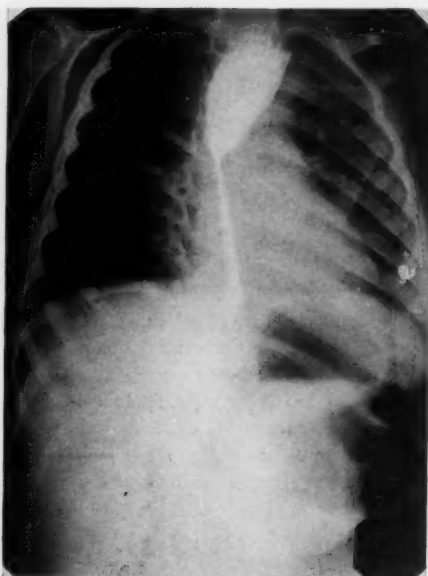


FIG. 7.—Roentgenogram taken approximately ten weeks after a lye burn, demonstrating practically a complete stricture of the lower third of the esophagus. There is considerable dilatation of the esophagus above the stricture.



FIG. 8.—Roentgenogram (lateral view) of the esophagus 34 months after treatment.

ESOPHAGEAL LESIONS

The child was discharged without treatment, but was readmitted one month later, during which interval she had lost 12 pounds in weight. At the time of readmission, she was able to take only fluids by mouth. July 30, 1935, a 6 Mm. esophagoscope was passed by Drs. A. E. Hammond and Wadsworth Warren. Just below the area of the cricopharyngeus there was a marked stricture. A No. 10 esophageal bougie was introduced, and the stricture dilated. Three months later, a 6 Mm. esophagoscope was again passed by Doctors Hammond and Warren, down below the cricoid to the upper esophageal stricture. It was impossible to get through the stricture further than about one-half inch with the smallest esophageal bougie available.

A gastrostomy was deemed advisable and the Stamm type of operation was performed October 30, 1935. On November 12, 1935, a 6 Mm. esophagoscope was passed down to the upper stricture. This was dilated with some difficulty by a No. 10 French bougie. Gradually the esophagoscope, together with the bougie, was passed down through to the lower esophagus. The No. 10 French bougie was then withdrawn, with the string positor, and the bougie was passed through into the stomach. With the aid of the esophagoscope in the stomach, the string was grasped and brought out through the abdominal opening. A heavy silk cord was then tied and withdrawn through the esophagus, inserted through the nares, and the loop completed. A week later, the esophagus was dilated, using a retrograde Tucker French No. 12 bougie. Some resistance was encountered. After four months (March 31, 1936) it was possible to dilate with a No. 20 French bougie, dilatation having been performed every two weeks. On July 21, 1936, it was possible to pass a No. 24 French bougie, and on September 15, 1936, retrograde esophageal dilatation with a Tucker No. 30 French bougie was accomplished. During 1937, the patient was dilated with a Tucker No. 30 French bougie every month, with no difficulty. At the time of the dilatation, November, 1937, she was told to return in three months. On April 19, 1938, a retrograde Tucker No. 30 French bougie was passed without difficulty (Fig. 8). It is planned at this time to close the gastrostomy, inasmuch as the patient has been taking solid food for the past year with no evidence of contracture of the former stricture.

RUPTURE OF THE THORACIC ESOPHAGUS

Case Report.—C. B., white, male, age 2, was admitted to the hospital August 6, 1935, with a history of having been well until July 30, 1935. The swallowing of a foreign body on that date was suspected. It was reported that previous to admission to the hospital, the child had eaten very little, and had vomited repeatedly since the onset of symptoms.

Physical Examination revealed a well nourished child, appearing dehydrated. Temperature, normal; pulse, 110; respirations, 24. The fontanelles were closed; pupils, equal, and reacted to light; ears and nose, negative; tongue, dry; and the throat, slightly injected, but no foreign body was seen. There was no rigidity of the neck, but on examination of the chest, an occasional coarse râle was heard over both bases. The heart borders were normal, and no murmurs were heard. The abdomen was soft; liver and spleen not palpable. A diagnosis of suspected foreign body in the esophagus was made. On August 7, 1935, the patient was esophagoscoped by Dr. A. E. Hammond, who found no evidence of a foreign body in the esophagus, but did see an ulcer approximately 1x1 cm. in the left lateral wall, about one inch above the cardia. The floor of the ulcer was necrotic and surrounded by a zone of hyperemia. After this examination, the child strained considerably and vomited. Approximately four hours later, there were signs of a left tension pneumothorax, which was aspirated, 400 cc. of air and 20 cc. of sero-sanguineous fluid being removed, with marked relief of the dyspnea. This finding of tension pneumothorax with fluid in the pleural cavity led us to suspect that the esophageal ulcer had perforated into the left pleural cavity.

Fluoroscopic examination, at 9:00 P.M., revealed that there was a total collapse of the left upper lobe, and a 50 per cent collapse of the left lower lobe, with marked displacement of the heart to the right. The child was placed in an oxygen tent with no

apparent improvement. Because of the increase in the severity of the tension pneumothorax and the inability to control it by aspiration, it was deemed advisable, at 11:00 P.M., to institute a closed catheter type of drainage under water. This resulted in marked relief of the dyspnea and cyanosis. Unknowingly, a student nurse gave the child milk to drink, and immediately the milk appeared in the drainage tube which had been placed in the left pleural cavity. This definitely proved that there was a perforation of the esophagus into the left pleural cavity. A Levine tube was inserted into the stomach for feeding purposes, putting the esophagus at rest. Fluoroscopic examination and roentgenologic examination, August 8, 1935, showed the drainage tube in the left pleural space, with no indication of free air or fluid in the left pleural cavity. The rectal temperature varied from 102° to 104° F. for two days following the rupture of the esophagus.

Roentgenologic examination, August 15, 1935, revealed the lung almost completely reexpanded. The Levine tube was used for feeding purposes until August 20, 1935, when fluids were given by mouth. The fluid which drained through the catheter showed no growth on culture. The tube was removed from the pleural cavity on August 26, 1935, and the child was discharged four days later.

Subsequent Course.—Fluoroscopic study, September 30, 1935, after a barium meal, showed that there was no interruption in the downward progress of the barium into the stomach. The lumen of the esophagus was reported small, but there was no abnormality in the contour of this structure at the site of the previously reported rupture. May 24, 1937, 21 months after the rupture, there were no symptoms referable to the esophagus or thoracic organs. Fluoroscopic examination after the administration of barium revealed no evidence of obstruction or stricture in the esophagus.

CONCLUSIONS

- (1) Various lesions of the esophagus coming to the attention of the surgeon have been briefly discussed and cases illustrating their management have been described.
- (2) The management of lesions of the esophagus requires close cooperation between the endoscopist, roentgenologist and surgeon.
- (3) The two stage procedure of esophagotomy carries a low mortality rate if performed at the optimum period.
- (4) Strictures of the esophagus demand careful treatment over an extended period in order to obtain satisfactory end-results.
- (5) Rupture of the thoracic esophagus demands early diagnosis and drainage of the pleural cavity.

REFERENCES

- ¹ Truesdale, Philemon E.: Oesophagotomy for Foreign Bodies in the Esophagus. *ANNALS OF SURGERY*, **80**, 375, 1924.
- ² Jackson, Chevalier, and Jackson, Chevalier L.: *Bronchoscopy, Esophagoscopy and Gastroscopy*. W. B. Saunders, 3rd Ed., 1934.
- ³ Pearse, Herman E., Jr.: The Operation for Perforations of the Cervical Esophagus. *Surg., Gynec. and Obstet.*, **56**, 1923, 1933.
- ⁴ Lahey, F. H.: Pulsion Esophageal Diverticulum. *J.A.M.A.*, **109**, 1414, 1937.
- ⁵ Graham, E. A., Singer, J. J., and Ballon, H. C.: *Surgical Diseases of the Chest*. Lea and Febiger, 1935.
- ⁶ Horsley, J. Shelton, and Bigger, Isaac A.: *Operative Surgery*. C. V. Mosby Co., 4th Ed., 1937.
- ⁷ Shallow, T. A.: Discussion of F. H. Lahey's paper. *J.A.M.A.*, **109**, 14-18, 1937.
- ⁸ Judd, E. S.: Esophageal Diverticula. *Arch. Surg.*, **1**, 38, 1920.
- ⁹ Penberthy, Grover C., and Benson, Clifford D.: Rupture of the Esophagus in a Child Two Years of Age; With Recovery. Submitted to *Surgery*, for publication, 1938.

LYMPHATIC SPREAD OF CARCINOMA OF THE RECTUM

RICHARD K. GILCHRIST, M.D., AND VERNON C. DAVID, M.D.

CHICAGO, ILL.

THE OPERABILITY and prognosis in patients with carcinoma of the rectum depend on the presence and extent of lymphatic metastases as well as on the degree of local extension of the tumor and the absence of blood-borne metastases to the liver, lungs, bones, brain, *etc.* The present study was undertaken in an effort to determine the incidence, extent and location of lymph node metastases and the extent of radical removal necessary to insure eradication of all involved nodes.

At first, this was done by a very careful dissection of the fresh, surgically removed specimen of carcinoma of the rectum. A full scale drawing was made, and the location of all lymph nodes was carefully noted on it. Between eight and 48 lymph nodes were found in each of 22 specimens examined by gross dissection. Nodes which were thought to be involved with carcinoma were labeled and circled with black ink on the drawing. All nodes were then sectioned and examined microscopically.

Recognition of involved nodes by palpation is difficult or impossible when they are small. Four hundred ninety-six nodes from these specimens were examined grossly to determine the presence of metastases. Of the 111 lymph nodes containing carcinoma, only 48 showed any gross change, even in cross-section of the gland. Gabriel, Dukes and Bussey¹ tried to determine the presence of lymph node metastases in 1,242 nodes found in operative specimens of carcinoma of the rectum. Of 337 nodes considered by them to have carcinoma grossly, only 132 were found to be involved when examined microscopically; and of 905 nodes considered to be free of carcinoma, 18 contained carcinoma when examined microscopically.

Technic of Examination.—Later, in order to more accurately study all of the lymph nodes, we developed the following technic for examination of surgically removed specimens of carcinoma of the rectum. The ligature on the superior hemorrhoidal artery of the fresh specimen is removed and a small cannula is tied into the artery. The specimen is then perfused with a warm 1 per cent citrate solution under a pressure of about 100 cm. of water. Ligatures on the smaller vessels are removed so that the blood can be washed out, and as soon as there is a free flow of clear fluid from any open vessel, it is ligated. After one to three hours of perfusion, the specimen is white except for the extrinsic muscles surrounding the anal orifice, and for any small areas where there may have been an extravasation of blood. Such areas can be cleared by very gentle manipulation. After the specimen has become almost entirely white, the artery is injected with red lead. This is done under moderate pressure, using a syringe. The vessel is then tied and the specimen is



FIG. 1.—Hosp. No. 34300: Gross specimen and photomicrograph. A photograph of the cleared, transparent preparation of this specimen shows the arterial tree as branching, black lines. The lymph nodes are represented by the spherical, dark areas seen throughout the tissue. The accompanying diagram shows the location of the lymph nodes. Those containing carcinoma are indicated by solid black dots. The normal lymph nodes are represented by hollow circles.

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placed in 10 per cent formalin solution for 24 hours. It is then changed every 24 hours from 50 to 70 to 95 per cent to absolute alcohol. If the fascia propria is untorn or if the tumor is thick, it is better to leave the specimen in alcohol for a longer time. The bowel is then opened along the antimesenteric border. This preserves the arterial tree and allows for an accurate localization of the tumor and its regional nodes. The specimen will now be completely white. It is mounted on any suitable frame with a few silk sutures and placed in methyl salicylate. After 24 to 48 hours the specimen becomes almost completely transparent. The red arterial tree serves as a very good landmark. The tumor, mucosa and peritoneum can all be easily recognized and the lymph nodes stand out as spherical masses of a slightly greater density than the surrounding tissue. Many bubbles will be found. These can be expressed by gentle pressure. The tissue can be handled with ease and safety if left beneath the surface of the methyl salicylate. Sometimes it takes five to seven days for all bubbles within the depth of the tissue to disappear (Fig. 1).

A full scale drawing of the bowel, arterial tree and tumor is then made. A section of the tumor is taken and placed in methyl salicylate. The lymph nodes can now be removed, each one can be labeled separately and its position charted exactly in its relation to the artery. These nodes are then placed in methyl salicylate and they are ready to imbed in paraffin and section and stain without being run through the usual alcohol and xylol solutions.

Specimens studied in this way have had 20 to 80 nodes per specimen. The average in 25 transparent specimens removed by the Miles type of abdominoperineal resection of the rectum was 52.1 nodes per specimen. Sixteen of the 22 specimens studied by gross dissection had lymph node metastases, and 16 of the 25 studied by the method of clearing had metastases, an average of 68.1 per cent of all specimens studied. Previous studies of lymph node involvement are summarized in Table I.

TABLE I
SUMMARY OF INCIDENCE OF LYMPH NODE INVOLVEMENT BY PREVIOUS AUTHORS

Author	Number of Specimens Studied	Number of Nodes per Specimen	Percentage with Metastases to Nodes
McVay, J. R. ²	100	6.23	47%
Wood, W. Q., and Wilkie, D. P. D. ³	100	11.23	51%
Westhus, H. ⁴	74 (cleared)	25.33	59%
Gabriel, W. B., Dukes, C., and Bussey, H. J. R. ¹	100	28	62%
Gilchrist, R. K., and David, V. C.....	22 (gross)	23.9	68.1%
	25 (cleared)	52.1	

Dukes¹ classification divides tumors into Grade A, where the tumor does not penetrate the bowel wall and does not have lymph node metastases; Grade B, where the tumor extends through the bowel wall and involves adjacent tissue but does not have lymph node metastases; and Grade C, where

there are metastases to lymph nodes. An analysis of the tumors studied in this series is given in Table II, which shows that tumors having lymph node

TABLE II

SUMMARY OF GRADINGS OF METASTASES ACCORDING TO THE CLASSIFICATIONS OF BRODERS AND DUKES

	32 Specimens With Metastases*	15 Specimens Without Metastases
Broders' Grades		
1.....	0	3
2.....	20	11
3.....	10	1
4.....	2	00
Dukes' Grades		
A.....	0	5
B.....	0	10
C.....	32	0
Number of involved nodes per specimen		
1.....	5	
2.....	5	
3.....	4	
Many.....	18	

* Specimens with metastases to high lying nodes, 19.

metastases tend to be of a higher grade classification, according to Broders' grading, than those without metastases. Eighteen of the 32 specimens having metastases had four or more nodes per specimen involved. Nineteen of the 32 had node involvement at or above the bifurcation of the superior hemorhoidal artery.

Tumors arising predominantly on the mesenteric border of the bowel seem to metastasize to the lymph nodes more frequently than do those arising on the antimesenteric border (Table III).

TABLE III

LOCATION OF TUMOR ON CIRCUMFERENCE OF THE BOWEL

	Mesenteric Border	Antimesenteric Border	Circular
32 specimens with metastases.....	17	12	3
15 specimens without metastases.....	7	7	1

The duration of symptoms seems to have less effect than one would suppose on the number of nodes involved in operable cases. Three patients who had had symptoms for four months or less had 22, 25 and 28 involved nodes, while five patients who had had symptoms 12 to 18 months had zero, two, three, two, and 43 involved nodes (Table IV).

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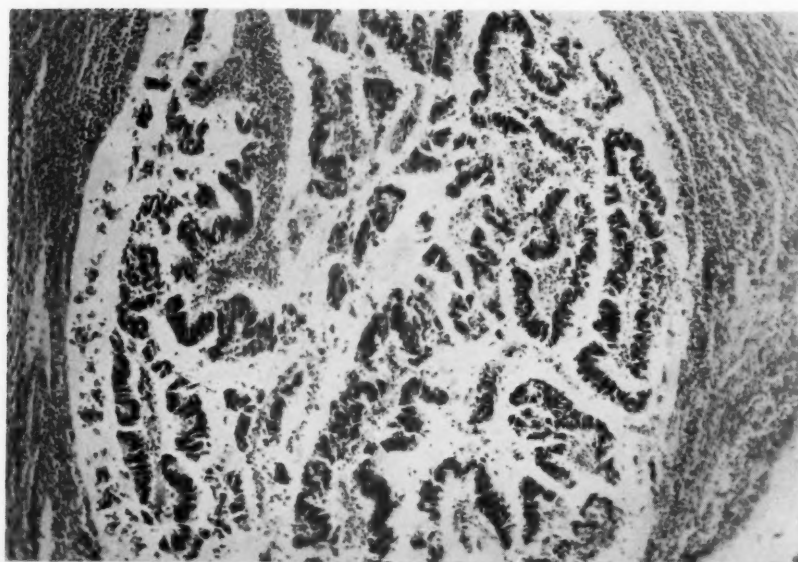
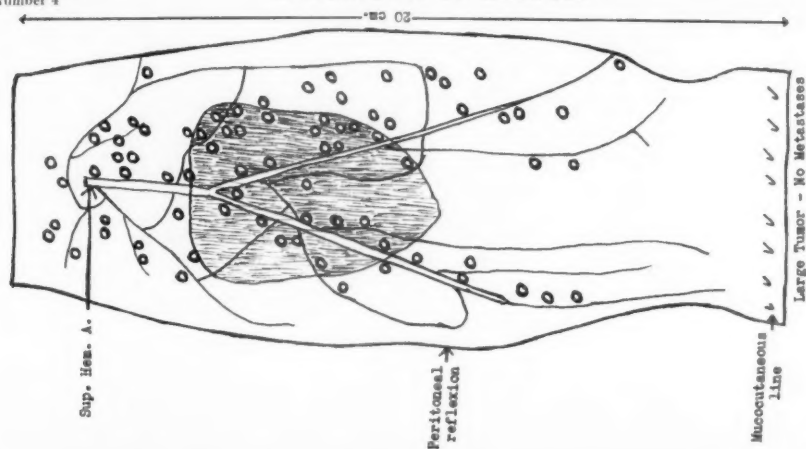


FIG. 2.—Hosp. No. 36463: Showing an instance of a large tumor without the occurrence of any metastases.

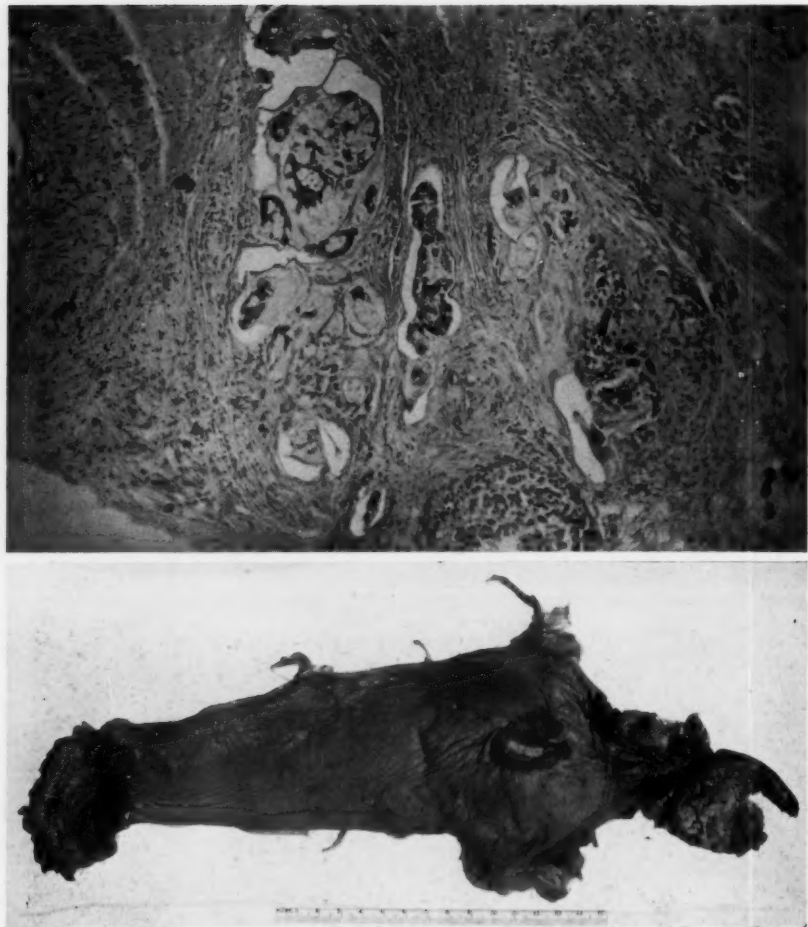
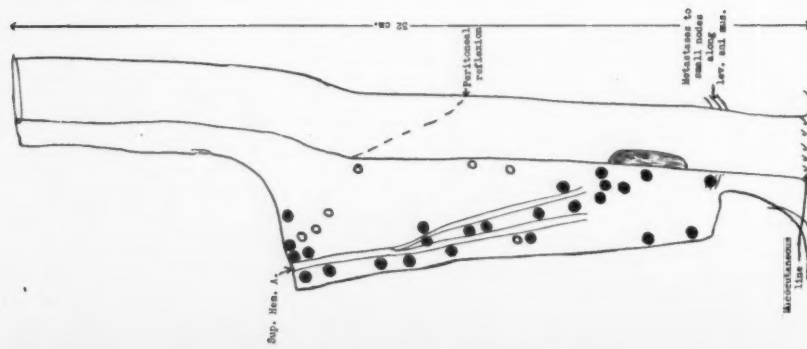


FIG. 3.—Hosp. No. 34434: Showing an instance of a small tumor with very extensive metastases.

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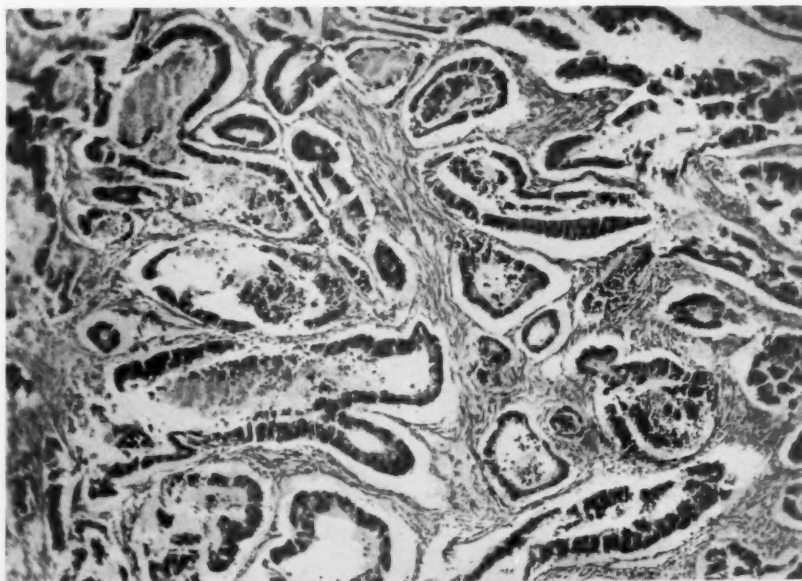
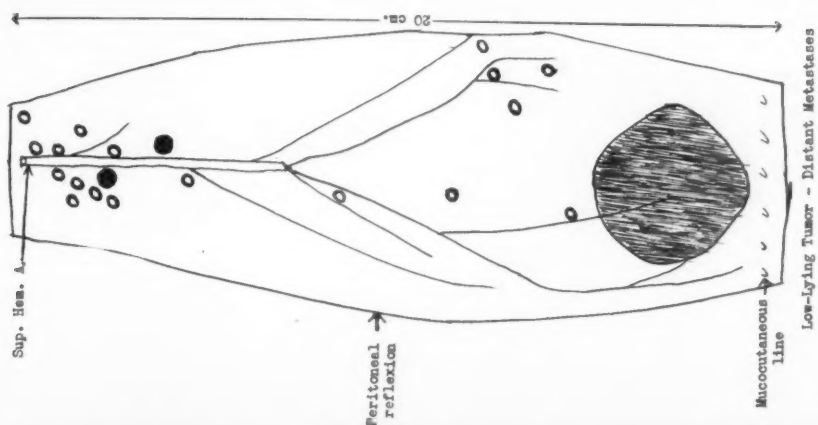


FIG. 4.—Hosp. No. 36255: Showing a low-lying tumor with distant metastases, without any apparent intervening involvement.

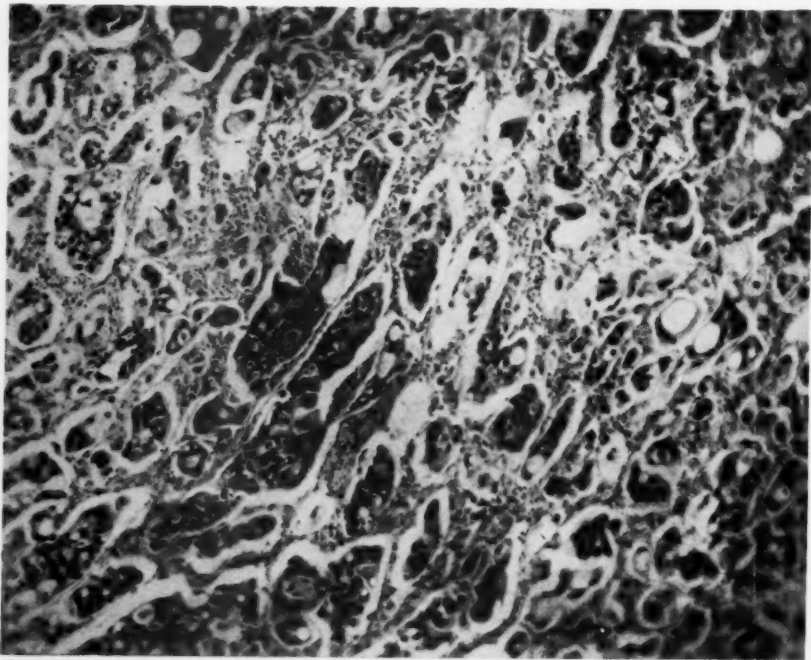
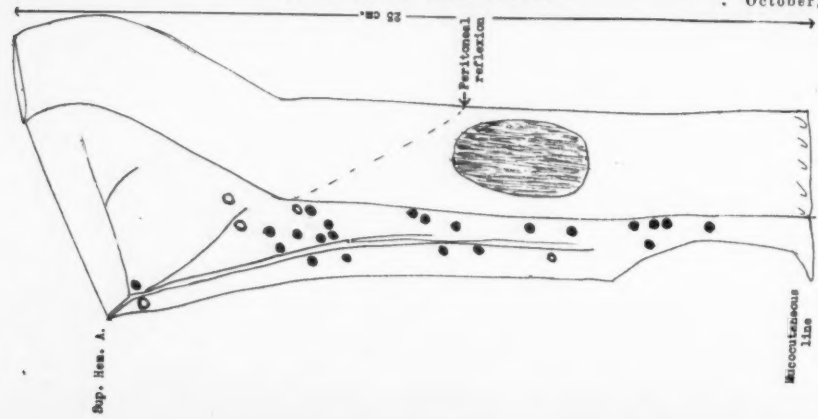


FIG. 5.—Hosp. No. 33779: Showing an instance of retrograde metastases. The lowest of the involved nodes being 4 cm. below the inferior edge of the tumor.

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TABLE IV

INCIDENCE OF METASTASES IN RELATION TO DURATION OF SYMPTOMS

Number of Nodes Involved per Specimen	Number of Cases with Symptoms 6 Months or Less	Number of Cases with Symptoms for More Than 6 Months
4 or more nodes.....	11	7
3 or less nodes.....	10	4
No nodes involved.....	6	9

The size of the tumor seems to have little relation to the number of nodes involved with metastases (Table V).

TABLE V

LYMPH NODE METASTASES IN RELATION TO THE AMOUNT OF CIRCUMFERENCE OF BOWEL INVOLVED

	More Than 75 Per Cent	Less Than 75 Per Cent
Specimens with 3 or less involved nodes.....	7	7
Specimens with 4 or more involved nodes.....	7	11
Specimens without metastases.....	8	7

Analysis of the specimens studied in this series has led us to the following conclusions:

(1) The size of the tumor is of little value in determining the presence or absence of lymph node metastases. Figure 2 (No. 36463) is an excellent example of this phenomenon. The patient, age 60, had had symptoms for eight months. He was very obese, which made radical removal difficult. The tumor involved at least 75 per cent of the circumference of the bowel. Eighty lymph nodes were removed from this specimen; all were normal. In contrast, Figure 3 shows the specimen from a patient, age 49, who had had symptoms for two months. The tumor involved about 20 per cent of the circumference of the bowel. The specimen was studied by gross dissection; 32 nodes were found, 25 of them contained metastases. Sixteen of the 25 involved nodes showed pathologic changes on sectioning, before fixation. There was one node found at the level of the levator ani muscle which was involved.

(2) Low-lying tumors may have metastases very high. Figure 4 shows the specimen of a patient, age 43, who had had symptoms for 16 months. The tumor involved about 65 per cent of the circumference of the bowel. Twenty nodes were found and the two solid black dots indicate the location of the lymphatic metastases.

(3) Where the upward lymph channels are blocked by metastases, there may be a retrograde metastasis downward as shown in Figure 5. The patient, age 65, had had symptoms for at least six months. The tumor involved about 65 per cent of the circumference of her bowel. Twenty-seven nodes were found by gross dissection, 22 of them having metastases. The lowest of the five nodes below the tumor was 4 cm. below the lowest edge of the tumor. One other case showed the same anatomic distribution.

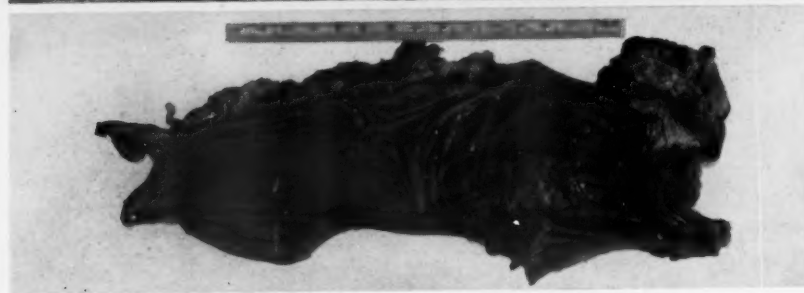
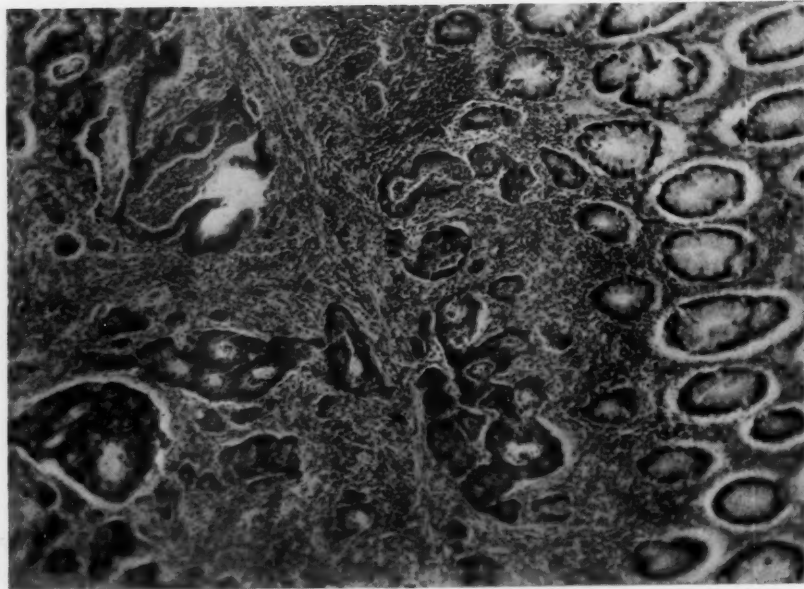
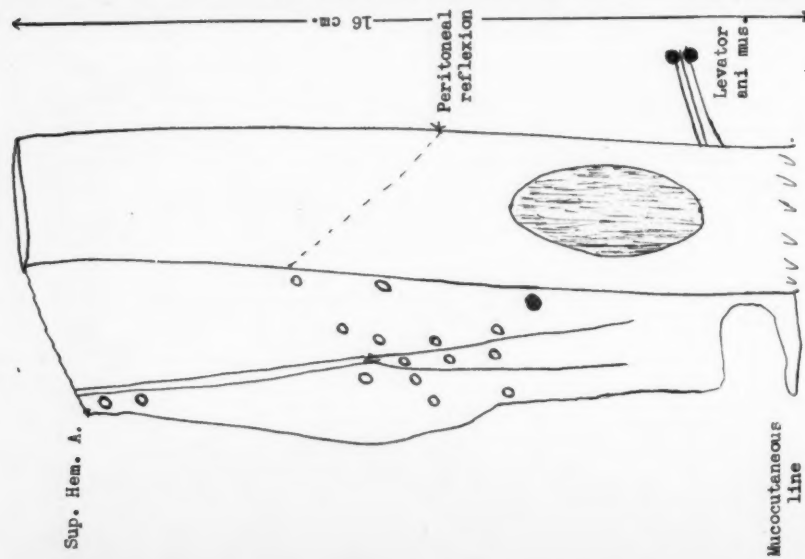


FIG. 6.—Hosp. No. 34446: Showing a double lymphatic drainage from a tumor situated at the level of the levator ani muscle, one node of the superior lymphatic drainage path and two nodes situated 2.5 cm. laterally to the muscularis of the bowel being found involved.

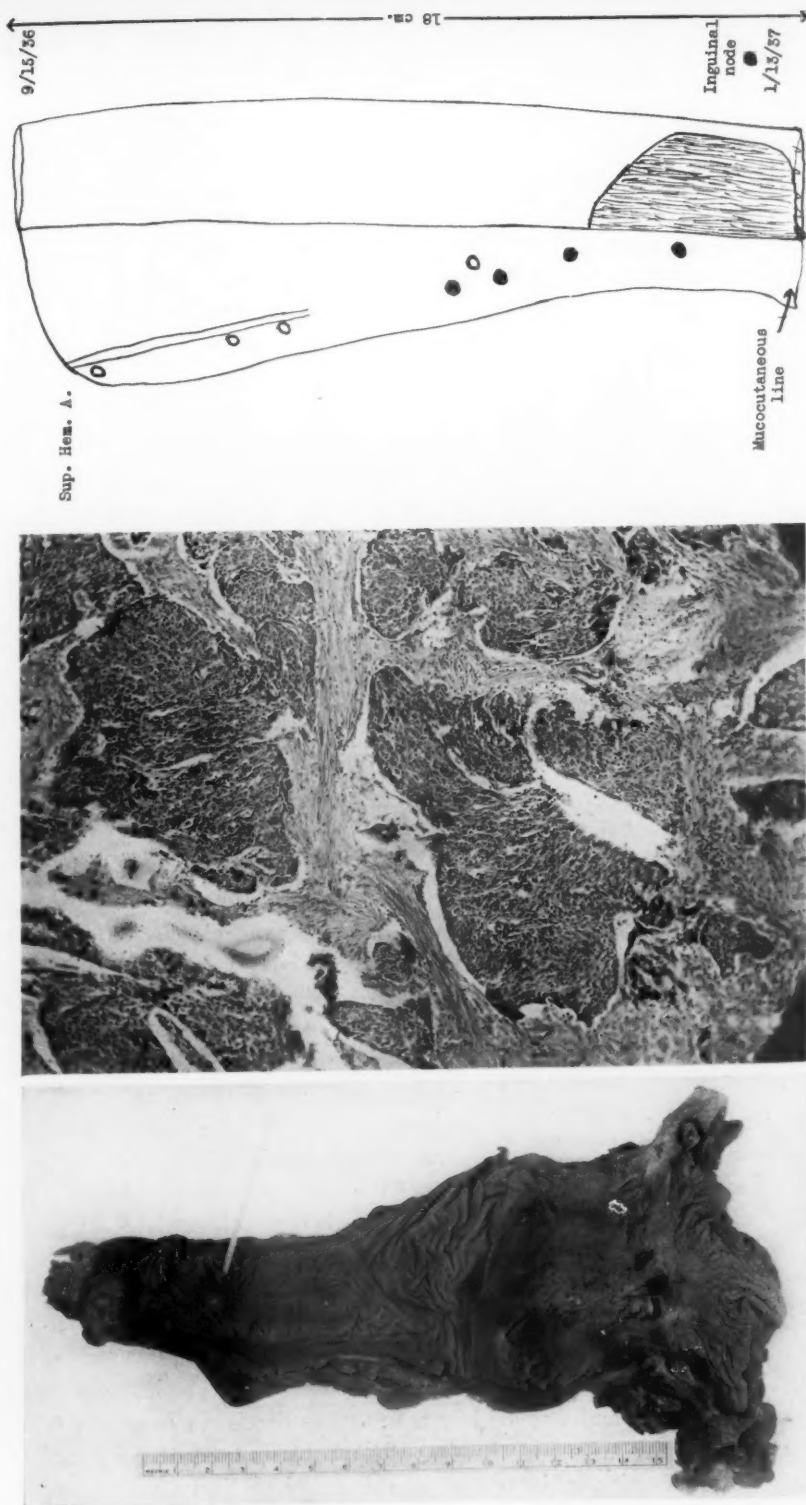


FIG. 7.—Hosp. No. 33289: Showing a double lymphatic involvement, resulting from an instance of a squamous cell carcinoma originating at the anal mucocutaneous junction; metastases having occurred in both the lymphatic nodes accompanying the superior hemorrhoidal artery and in the inguinal nodes.

(4) Where the tumor is found at the level of the levator ani muscle, there is a double lymphatic drainage: the more common is upward along the superior hemorrhoidal artery, the other direction is laterally along the superior surface of the levator ani muscle. Lymph node metastases are found here. Four such instances occurred in this series. One has been cited above (Fig. 5.—Hosp. No. 33779). The specimen illustrated in Figure 6 is from a patient, age 53, who, while in the hospital recovering from fractures of both

legs, developed rectal bleeding. Six weeks later he was operated upon for carcinoma of the rectum. The tumor involved 50 per cent of the circumference of the bowel. Eighteen nodes were found by gross dissection, three of them being involved by carcinoma. The two along the levator ani muscle were 2.5 cm. lateral to the muscularis of the bowel.

(5) Squamous cell carcinomata which involve the mucosa may have a double lymphatic involvement. Figure 7 illustrates the specimen removed from a patient, age 60, who had had symptoms for six weeks. The tumor originated at the mucocutaneous line and extended upward for 5 cm. involving the mucosa but without producing any ulceration. There had never been any bleeding. It involved about 80 per cent of the circumference of the bowel. Eight nodes were found by gross dissection; four of them along the course of the superior hemorrhoidal artery, above the tumor, were involved. Four months later the inguinal nodes were removed and one of them showed metastatic involvement.

(6) Postmortem examination shows that radical removal, with resection of

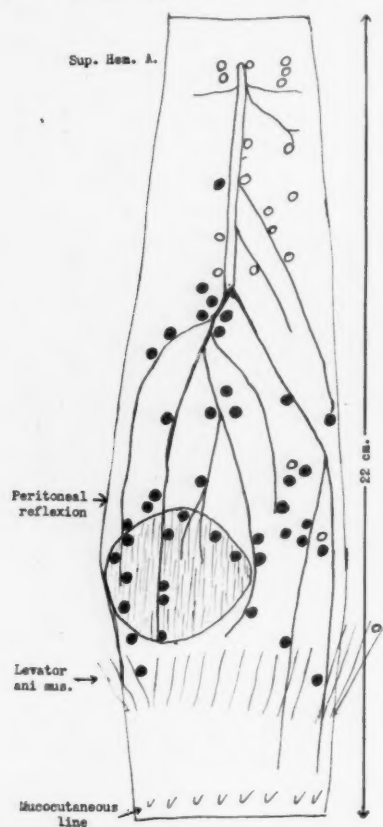


FIG. 8.—Hosp. No. 36303: Showing the extent, both superiorly and inferiorly, that metastases can occur, and how essential a Miles type of combined abdominoperineal resection is, if one wishes to eradicate them.

the superior hemorrhoidal artery as high as possible and wide resection of the levator ani muscles, is necessary in order to give the best chance of permanent cure. Figure 8 illustrates the specimen removed from a patient, age 64, who had had symptoms for 12 months. The tumor was ulcerating, and involved 60 per cent of the circumference of the bowel. It had penetrated all coats of the bowel and was slightly adherent to the fascia propria. Nodes were palpable in the hollow of the sacrum. A Miles type of combined abdominoperineal resection of the rectum was performed. Sixty-two nodes were

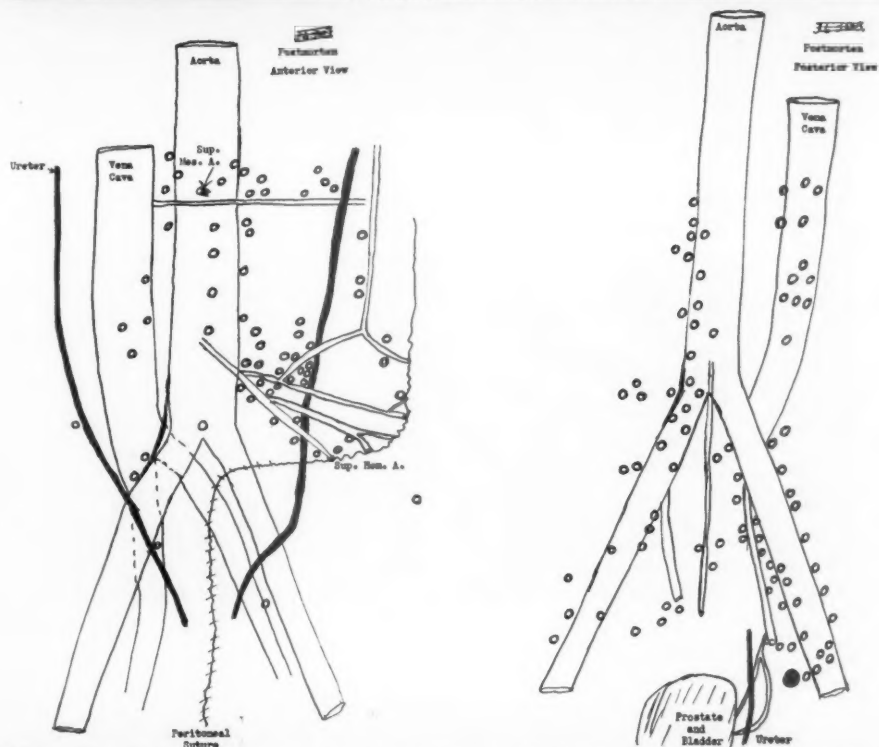
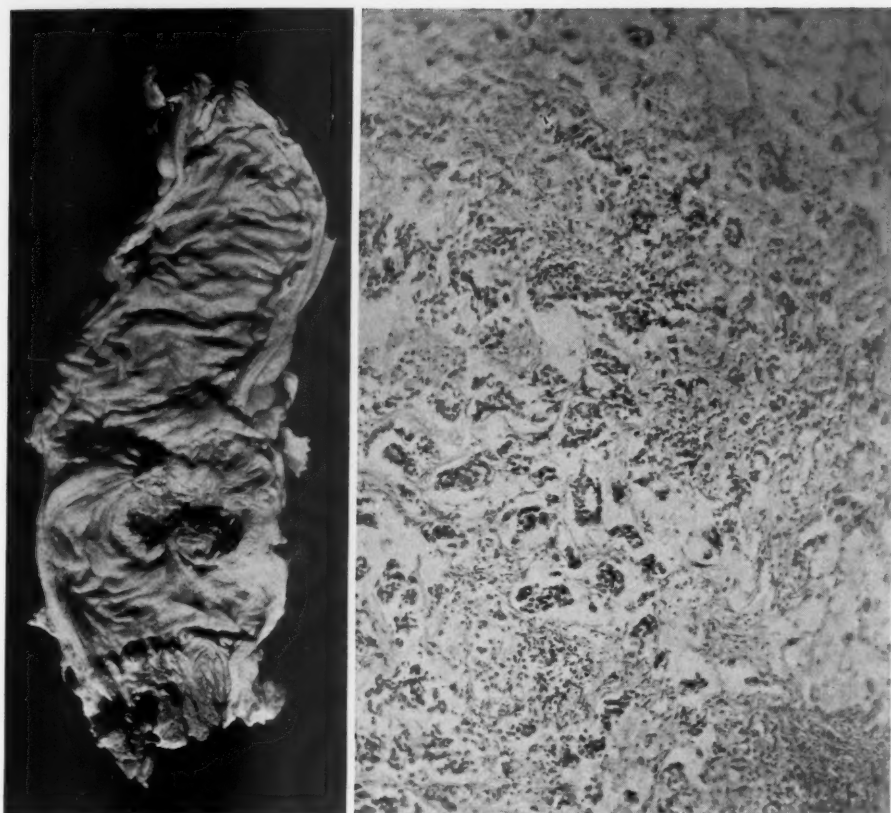


FIG. 9.—Hosp. No. 36303: Autopsy. Anterior and posterior sketches showing the lymph node distribution.

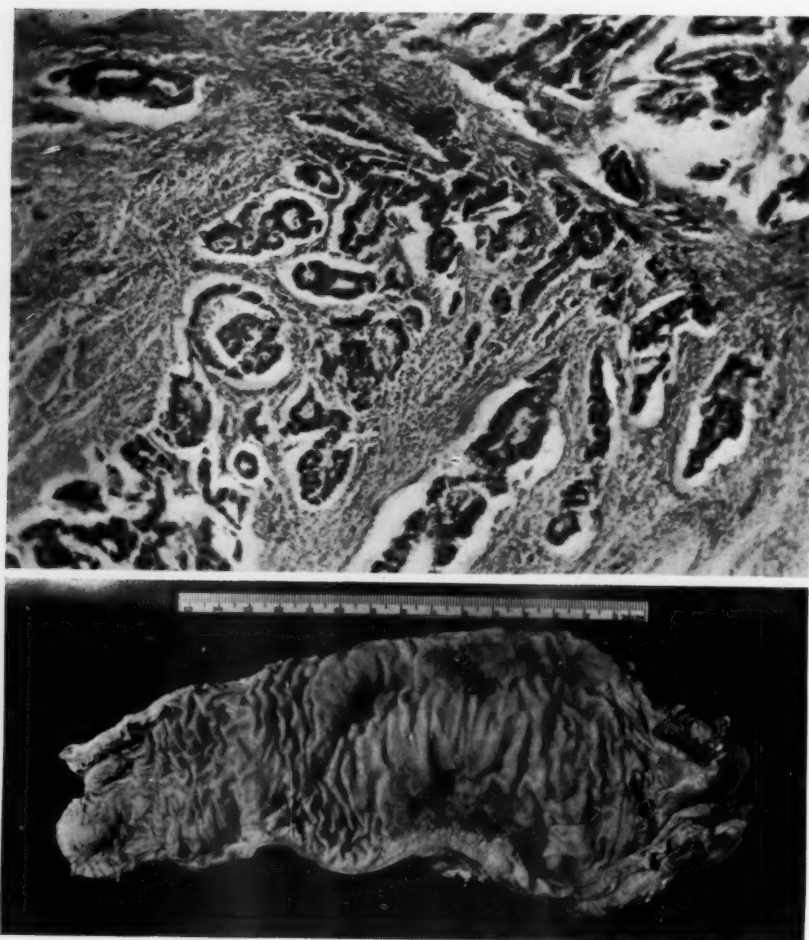
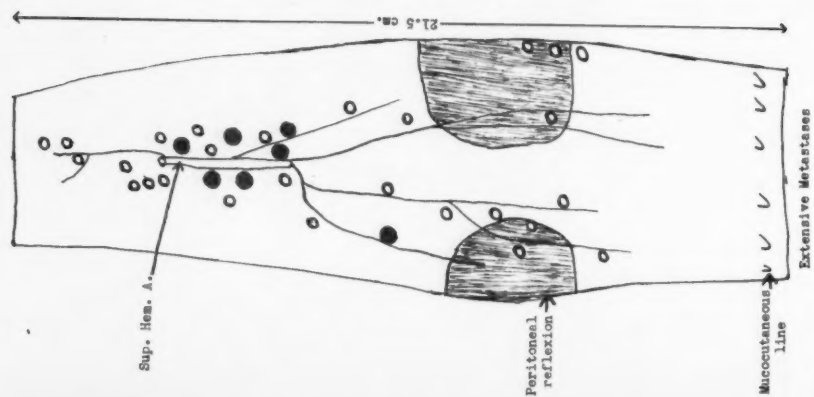


FIG. 10.—Hosp. No. 36642: Showing the high distribution of the involved lymphatic nodes.

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found; 43 of them showed metastases. The highest node involved was only 3 cm. below the point of ligation of the superior hemorrhoidal artery. He had an uneventful postoperative course until the eighth postoperative day,

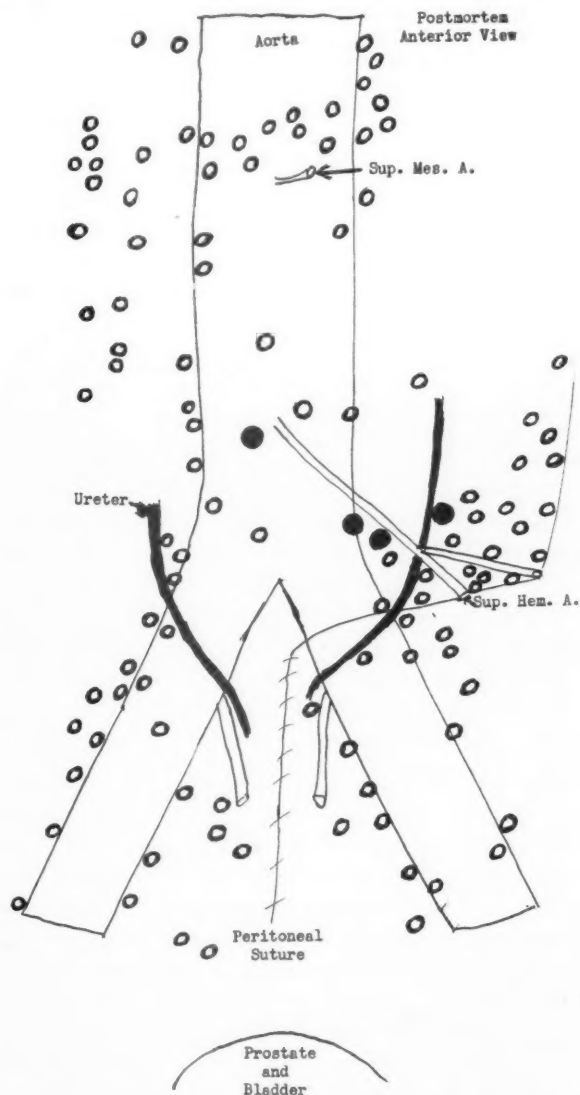


FIG. 11.—Hosp. No. 36642: *Autopsy*. Sketch showing the lymphatic node distribution; and the presence of involved nodes remaining postoperatively, which would have been impossible of surgical removal.

when he suffered a massive pulmonary embolus and died. The two diagrams of the postmortem preparation show the location of the 160 nodes which were examined. The highest node was 3 cm. above the point of origin of the superior mesenteric artery, and the lowest was at the inferior border of the prostate, as far distal as it is possible to cut the arteries from within the abdo-

men. In spite of the extensive lymphatic involvement in the operative specimen, there were no metastases above the point of resection. The one node involved was about 1 cm. lateral to the widest point of resection, along the levator ani muscle (Fig. 9).

Figure 10 illustrates the specimen removed from a very thin and feeble patient, age 72, who had had symptoms for six months. The tumor involved 65 per cent of the circumference of the bowel. There were a number of enlarged nodes high up, and because of a peculiar congenital peritoneal anomaly, the superior hemorrhoidal artery could not be resected as high as it frequently is. Thirty-five nodes were found in the operative specimen, seven of them contained metastases. He died of an aspiration bronchopneumonia. The diagram of the postmortem preparation shows the location of the 111 nodes which were studied. Four nodes were found to be involved, demonstrating that a complete removal was not possible in this case.

CONCLUSIONS

Prognosis based on the spread of the tumor into the lymph nodes is inaccurate unless a careful dissection of the specimen is made.

When a very careful search for lymph nodes is made, either by gross dissection or in the cleared specimen, about 68 per cent of all operatively removed specimens of carcinoma of the rectum will have metastases to lymph nodes, and the more nodes examined, the greater the number per specimen which will show carcinoma.

Recognition of involved nodes by palpation is difficult or impossible where the nodes are small, since only 48 of 111 lymph nodes containing carcinoma showed any gross change, even in cross-section of the node.

Tumors which are questionably operable because of the large size of the tumor and because of obesity in the patient may have no lymph node metastases.

Small tumors may have very extensive lymphatic node metastases.

Tumors having lymph node metastases tend to be of a higher classification, according to Broders' grading, than those without metastases.

A high ligation and division of the superior hemorrhoidal artery, and of the lymphatics accompanying it, is desirable wherever possible, since low-lying tumors may have high-lying metastases.

Where there is gross enlargement of the high-lying nodes, with lymph blockade, there may be retrograde metastasis below the tumor.

Where the tumor is near the level of the levator ani muscles, those muscles should be resected as widely as possible, since metastasis along them seems to be common.

Squamous cell carcinomata metastasize upward along the course of the superior hemorrhoidal artery, as well as laterally to the inguinal lymph nodes, when the mucosa is involved. Therefore, a radical resection of the rectum should be performed whenever a squamous cell carcinoma has involved the mucosa.

The Miles type of operation seems the ideal one from the standpoint of wide removal of the lymphatic node bearing area.

REFERENCES

- ¹ Gabriel, W. B., Dukes, C., and Bussey, H. J. R.: *Brit. J. Surg.*, **23**, 395, 1935.
- ² McVay, J. R.: *ANNALS OF SURGERY*, **76**, 755, 1922.
- ³ Wood, W. Q., and Wilkie, D. P. D.: *Edinburgh, M. J.*, **40**, No. 7, 328, 1933.
- ⁴ Westhus, H.: *Die Pathologisch-anatomischen Grundlagen der Chirurgie des Rektumkarzinoms*, 78, 1934.

DISCUSSION.—DR. VERNON C. DAVID (Chicago, Ill.): This is rather discouraging information. It seems to me practically to indicate that the more radical procedure attempted, and particularly the higher ligation of the superior hemorrhoidal artery we do, the more likely we are to be above nodes which are involved, no matter what the position of the tumor in the bowel may be.

We now are ligating the superior hemorrhoidal artery just distal to the first sigmoid branch, which usually, in the average patient, is about two to two and one-third inches above the promontory of the sacrum.

One other fact that seems to me of practical importance is that where nodes are palpable (and this work shows that they are not all palpable that are involved), and presumably involved, there seems to occur a blockade of the normal spread of the disease along the vessels, and detours are made in which nodes may occur in the retrograde lymphatic group as well as the lateral group; and therefore it has seemed to us that in cases of that type, we should start our dissection laterally at the level or plane of the ureters and work toward the bowel.

I feel very sure that there are other lymphatics than those described so adequately by Miles that traverse the well known routes described by him. I am sure that all of us have seen carcinomata on the anterior wall of the bowel of women where, without much adherence to the rectovaginal septum nodules have been found in the vaginal mucosa.

About a month and a half ago, we saw a woman for the first time with an otherwise operable carcinoma, the size of a dollar, on the lateral side of the bowel, not attached at all to the rectovaginal septum, that had a half dozen metastases in the vaginal mucosa. No others could be found. Therefore, that indicates that there may be other routes that are certainly not favorable, but I think they occur.

DR. HARVEY B. STONE (Baltimore, Md.): I think that both Doctors David and Gilchrist certainly deserve a great deal of respect and admiration for the very painstaking and laborious piece of research which they have carried out. Doctor Gilchrist, out of consideration for our patience, did not describe the very laborious, time-consuming technic required to clear these specimens, to draw charts of each one of them, to label each node found, and then to correlate the microscopic study of those individual nodes with their position on the anatomic charts. It is really an overwhelming piece of work and certainly throws valuable and important light on a very practical and alive surgical problem.

Many of the observations which they recorded, of course, have been foreshadowed by the work of Miles and others, but it seems to me that there are certain new points or points of renewed emphasis that are well worth repeating in this brief commentary.

In the first place, I think that we have all realized that there was no close

correlation between the size of the primary growths and the metastatic involvement, but my own reaching of that conclusion had not been based upon the finding of involved lymphatic nodes but rather upon the fact that I think all of us must occasionally have observed: that sometimes a quite small primary growth in the rectum is accompanied by palpable nodules in the liver.

I had always explained that to myself as an accidental invasion of the blood vascular system with direct embolic transportation to the liver, but now the work just presented shows that in addition to these presumably accidental metastases, one must routinely expect the possibility at least of widespread lymphatic extension from comparatively small primary growths.

Another thing that amazed me was the number of nodes found in these specimens. I had, from my own dissection and observations of specimens removed, concluded that an average of perhaps 15 or 18 nodes was the normal equipment of lymphatic apparatus in such specimens, and it is rather surprising to learn that, in this bulk of material, an average of more than 52 nodes was found per specimen.

Furthermore, I think that the important conclusion derivable from this entire work is the light that it throws on the still persisting controversy between the Miles type operation and that of Lockhart-Mummery or some sleeve-type of resection, in which either an attack from below or an effort to preserve the sphincter muscle is the purpose of the operation.

I do not believe it is possible, in the face of the evidence here presented, to feel that one has done everything possible to perform a radical removal of carcinoma of the rectum unless the Miles type operation is adopted as a standard procedure. It seems to me that with this evidence, no one can feel that a perineal resection by the Lockhart-Mummery or any other type of attack from below can give any assurance whatever that the metastatic nodular involvement has been removed.

I think another important observation made is this, that we have all been taught, and properly so, that because nodes were palpable, they were not necessarily involved. Now, there is an even more important corollary in these observations, which is that because nodes are not palpable, is no proof that they are not involved.

DR. RALPH COLP (New York, N. Y.): I hesitate to show our series, because I think as you glance at the slides you will come to the conclusion that we probably have not been radical enough in our abdominoperineal resections, because the number of nodes which we found were far less, and certainly the number of cases which presented metastasis were fewer.

We originally started to dissect these nodes by the method advocated by McVay, Wood and Wilkie, but soon found we were missing a certain number, so we decided we would employ the method advocated by Westhue, which evidently is not as good as the one which Doctor David presented. The Westhue method is based upon a fixation in Kaiserling I solution, then dehydration in alcohol, and then finally cleared in methyl salicylate solution.

Up to date, Dr. S. H. Klein in our laboratory, under Doctor Klemperer's direction, has examined 18 cases, which I would like to present.

Table I details the findings in these 18 cases, all but two of which were operated upon in two stages by the Lahey procedure. We found some cases with only nine, 14, and 17 nodes, and others with 30, 34 and 45. It is rather interesting that the case in which the fewest nodes were present was one in which a perineal resection was performed because we felt that the patient would not tolerate an abdominoperineal resection, and it is quite obvious that a great number of nodes were left behind. In another case in which only nine

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TABLE I

CARCINOMA OF THE RECTUM AND RECTOSIGMOID*

Case No.	Lymph Nodes Found	Lymph Nodes Containing Metastatic Carcinoma
55620.....	34	5
11239.....	9	0
57590.....	11	0
57702.....	45	0
55869.....	30	0
59575.....	17	4
55802.....	14	0
51747.....	19	7
8260.....	4 (Hartmann operation)	0
58428.....	14	0
53763.....	15	0
57763.....	1 (Jones perineal resection)	0
59044.....	13	0
53809.....	28	0
57618.....	3	0
11679.....	22	0
51915.....	9	0
56108.....	16	0

* The two stage Lahey procedure was performed in all cases unless otherwise specified.

nodes were found, a Hartmann type of operation was performed, in which the lower rectal segment was left in place.

Another interesting point in this series is the paucity of metastases. In one case in which 34 nodes were found, only five were involved. In other cases, there were 17 with four, 19 with seven, and 22 and 28 nodes without metastases. In spite of the fact that all of these nodes were very carefully examined, no metastases were found.

I agree with Doctor David that very often a retrograde metastasis may be present due to the plugging of the lymphatics, and in this series, two such cases were found. Westhue, in his report, stated that in 74 cases he found only one instance in which there were retrograde metastases and felt this could be discarded as having no clinical significance. However, in this series in which only three cases showed metastases, two of them showed evidence of retrograde metastases.

Figure 1 illustrates the nodes found in one case. One will notice that some of these nodes were found distal to the tumor, and one node was at a distance of 5 cm. In the second case which we had, one of these nodes was found at a distance of 3 cm.

I think if these retrograde metastases can happen in a small series of cases, such as we have reported here, it certainly emphasizes, again, that the only

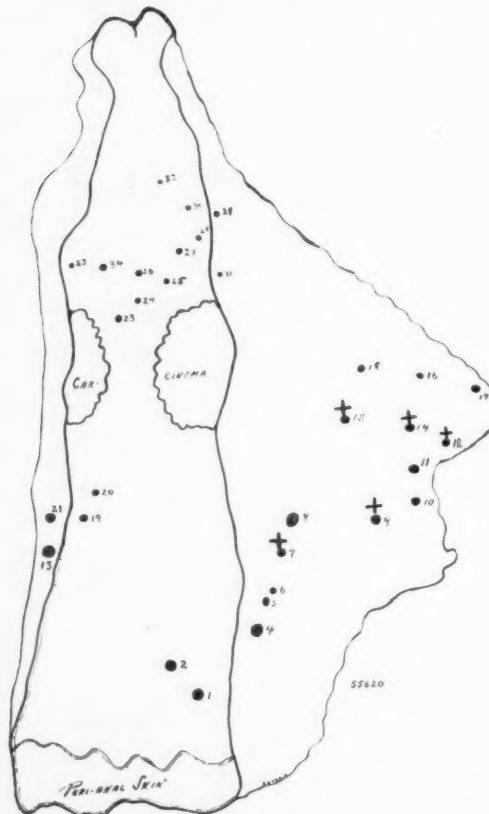


FIG. 1.—Schematic drawing of resected rectum and rectosigmoid. The numbered dots represent the regional lymph nodes. The nodes marked with plus signs exhibited metastatic carcinomatous involvement. Note that the positive nodes are situated distal to the primary growth.

type of operation which should be performed is a radical abdominoperineal resection, extending rather than some conservative types of procedure, in which an attempt is made to preserve the sphincters.

DR. EDWARD ARCHIBALD (Montreal, Canada): This paper of Doctors David and Gilchrist forms a welcome addition to our knowledge of the spread in the lymph nodes in cases of rectal cancer, and one naturally asks oneself whether their findings modify in any way the view generally held in this country that Miles' abdominoperineal operation is superior, in ultimate results, to the Lockhart-Mummery resection of the rectum from the perineal side. In the paper under discussion I noticed one thing particularly: that, although the lymph nodes adjacent to the cancer might be found free of disease, a node situated farther up might be involved. Such a fact tends to support the more extensive removal of the Miles operation. There remains, of course, the consideration of the question of operative mortality, and I think we must count it true that the Lockhart-Mummery will always show a better mortality rate after operation than the Miles procedure.

One other point in the present paper draws my attention—the relative infrequency of a spread of the lymph node extension downwards, that is, toward the anus; and this brings up once more, at any rate in the case of cancer situated three inches or more above the anal passage, the question of saving the sphincter. In 1907¹ I devised an operation which I hoped would combine the advantages of a complete block dissection of rectal cancer through the abdomen, with the preservation of the sphincter. It involved the mobilization of the descending colon and at times even of the splenic flexure and the section of the sigmoid mesentery far back, at the peritoneal reflection, so as to preserve the vascular supply in the arcades. The mobilized sigmoid was brought down and sutured to the anal stump of the rectum. I finally gave up the operation because it was difficult to be sure of the maintenance of arterial circulation in the sigmoid mesentery under the operative conditions of those days, when we did not know how to prevent operative shock, and patients such as these often came off the table with a very low blood pressure, lasting for hours, and a consequent imperfect circulation in the sigmoid stump. Injection experiments in the cadaver, it is true, had demonstrated the patency of the vessels in the sigmoid segment, when brought down without tension to the anal stump, but the injection experiments had not been made at low pressures; had, indeed, been made probably under a pressure far higher than even normal blood pressure, that is, with a syringe. The result, clinically, was too often necrosis of the sigmoid stump, failure of union, and ultimate obstruction from scar. During the past few years I have often thought that one might perhaps, under improved operative conditions as regards blood pressure, return to this operation and again make the attempt to save the sphincter. But two considerations still tend to block that path, at any rate still offer obstructions that must be surmounted. Of these, the first is the difficulty of securing primary healing of the anastomosis between the sigmoid and anal stumps, not only because of the possible risk of imperfect circulation but also because of the risk of infection. Our modern methods of aseptic bowel suture after resection are hardly feasible in the depths of the pelvis, whether one tries it from the abdomen or from the sacral side. And secondly, modern apparatus has succeeded in making the abdominal permanent colostomy very much less of a trial to the patient than in earlier days. There are still patients, and not a few, who say before operation that they would rather die than have a permanent colostomy. On the other hand, there does exist the diametrically opposite view; and, if you will allow me to conclude on a somewhat lighter note, I would quote the remark of a patient, recorded by one of our Paris colleagues years ago, concerning the annoyance of the artificial anus and the philosophic resignation which that was supposed to demand. This patient, finding that her colostomy was working decidedly well, and that she was not annoyed to anything like the degree which she had expected, said to her surgeon as he paid the final visit: "Doctor, I don't see why the good Lord didn't put our behinds in front; it's much more convenient!" If all of our patients were able to adopt the mental attitude illustrated in this French lady's observation, the chief opposition to the radical operation, with its permanent colostomy, would be overcome. Nevertheless, it is certain that many perfectly reasonable people will continue to view an artificial anus as one of the great trials of the flesh. In this sense I still think that there will occur a swing back to some method of preserving the sphincter, when the cancer is

situated not too near to the anal passage. Such methods, of course, have been devised and published, but at present they seem to be relegated to the lumber-loft, in favor of the abdominoperineal and perineal methods.

REFERENCE

- ¹ Operative Treatment of Cancer of the Rectum. J.A.M.A., 573-579, February 22, 1908.

ARTERIOVENOUS ANEURYSMS

MONT R. REID, M.D., AND JOHNSON MCGUIRE, M.D.

CINCINNATI, OHIO

FROM THE DEPARTMENT OF SURGERY AND THE DEPARTMENT OF INTERNAL MEDICINE OF THE COLLEGE OF MEDICINE, UNIVERSITY OF CINCINNATI, AND THE CINCINNATI GENERAL HOSPITAL, CINCINNATI, OHIO

IN 1925, Reid¹ published a series of four papers under the general title of "Studies on Abnormal Arteriovenous Communications, Acquired and Congenital." In these articles the literature of the subject was rather extensively reviewed. He put forward the thesis that there was no essential difference, except in the size and number of arteriovenous fistulae, between angiomata, cirroid aneurysms and arteriovenous aneurysms; and this view has been rather generally confirmed.

Thirty-three cases were reported and the clinical studies of them, together with laboratory investigation, formed the basis for certain remarks concerning the effects upon the body of abnormal arteriovenous communications, and their treatment. At that time the author realized that there were many matters in connection with this subject which had not been solved; that further investigations of it would yield important physiologic, pathologic and therapeutic observations. This has not only proven to be true, as witnessed by the important contributions upon the subject, but has led to by-paths which give promise of yielding important observations concerning conditions which are more frequently encountered than are abnormal arteriovenous communications. We have in mind cardiac disabilities, especially aortic insufficiency and cardiac failure, the state of the capillary bed, the normal absence of capillaries in certain parts of the human body and of other animals,² blood volume, circulation time, *etc.* Indeed, rarely has the investigation of such an infrequent clinical condition been so fruitful of important collateral contributions. This field of investigation seems limitless, for new problems always present themselves, and there are still many old ones which have not been solved.

The purpose of this report is to present another series of 30 cases (12 in detail), to discuss our clinical observations and surgical procedures and to supplement, whenever pertinent, these clinical studies from observations made in our laboratory of experimental surgery.

In this paper are included all the cases we have had since Reid¹ published his series of 33 cases, in 1925. Several of this series have been published previously in considerable detail and are included here only in the synopsis of cases (Table I). Where this has been done full reference is given to publications in which complete details may be found. They are included here again for two reasons: First, we wished to use data from them in our general discussion of the subject of abnormal arteriovenous communications; second, later observations on these cases may be of interest to someone. None of the 12 cases reported in detail in this paper has ever been published before.

Case 13.—Massachusetts General Hospital No. 29001 and No. 29001 R.A.: This patient was first admitted to the hospital May 5, 1931. At that time he was 31 years old. At about the age of 15 he injured his right leg with a hand sickle. The point of the blade entered the anterior surface of the leg lateral to the tibial crest and about two inches below the tibial tubercle. There was profuse bleeding which was controlled by a tourniquet until the wound was sutured. During the following years there was no disability, but the patient noticed that the right leg was a little larger and possibly longer than the left. There was no shortness of breath, even when rowing at college or when skiing.

The patient first came for examination because of the accidental discovery of a tumor, thrill and bruit in the region of the old sickle scar, when he was examined and treated for a sprained ankle. Shortly after this he came to Dr. A. W. Allen of Boston for the treatment of an arteriovenous aneurysm. On his first admission to the hospital moderate varicose veins were noted; the right calf measured 38 cm. and the left 35½ cm.; heart sounds were normal; blood pressure 140/80. Maximum pulsation, thrill and bruit were noted in the popliteal space, but the bruit could be heard over the entire femoral artery and its branches, as well as all about the knee and just below it.

Operation.—May 8, 1931: Doctor Allen ligated and divided a large artery, low in the popliteal space, which he thought to be the posterior tibial. The vein was not disturbed and great care was taken not to injure the posterior tibial nerve. The actual fistula was thought to be lower in the leg, but inasmuch as the ligation of the artery caused the thrill and bruit to disappear, no attempt was made to close it. After the completion of the operation, a pulse could be felt in the anterior tibial artery.

Two weeks after this operation a "toe-drop" developed and an area of anesthesia on the dorsum of the foot and ankle could be demonstrated. Some reddened areas just above the ankle were noted and they soon developed into a chronic ulcer which would not heal and occasionally bled profusely. A soft bruit and later a thrill made their reappearance in the region of the old stab wound.

During the year following this first operation the signs of an arteriovenous aneurysm became progressively more pronounced until they were almost as evident as before the operation. The patient wore a brace with a right-angle ankle stop for his partial peroneal nerve palsy; the ulcer refused to heal and occasionally bled. During this year it was necessary for him to use crutches all of the time. In October of 1931, Dr. F. R. Ober noted a barely palpable pulse in the dorsalis pedis artery and a good pulse in the posterior tibial. In addition to confirming the diagnosis of partial paralysis of the peroneal nerve, he observed that a shortened Achilles tendon limited dorsal flexion of the ankle about 20 degrees.

About a year after the first operation Doctors Allen and Reid saw this patient together. The circulation of the foot and ankle was definitely impaired, although feeble pedal pulses could be felt. The chronic ulcer above the ankle was about the size of a silver dollar, grayish, cyanotic in appearance and exhibited no evidences of healing. In the skin about the knee and upper leg the veins were so numerous and distended as to give an angiomatous appearance. Anteriorly at the site of the stab a forceful pulsation could be felt. Here and all about the knee was a distinct thrill. The loud continuous bruit, with systolic intensification, could be heard, with diminishing intensity, up to the groin and down to the ankle. The femoral and popliteal arteries appeared to be two or three times their normal size. Occlusion of the arteriovenous fistula by direct pressure caused, after a very short time, a definite increase in the volume of the pedal pulses and a slowing of eight to ten beats in the heart rate. The action of the heart was regular and no enlargement was demonstrated by physical examination. At the time of these studies (May, 1932) an extirpation of the fistula was advised.

Second Operation.—August 9, 1932: Doctors Allen and Reid. The duration of the operation (five hours) bespeaks the tediousness of the procedure and the great vascularity brought about by the secondary angiomatous condition of the tissues, as well as the

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technical difficulty of attacking directly an arteriovenous fistula situated at the bifurcation of the popliteal artery. Figures 1 and 2 make unnecessary an extended description of the operative procedure. The approach, as in the first operation, was again made



FIG. 1.—Case 13: Arteriovenous aneurysm between popliteal vessels at the level of origin of the anterior tibial vessels. The artery was ligated 15 months before this operation. The peroneal nerve is infiltrated with large blood vessels which caused its partial paralysis.

through the popliteal space. The popliteal artery was about three times its normal size and very thin-walled; it was completely occluded about one inch above its bifurcation. The enormous popliteal vein was intact and through its wall could be seen the play of mixing arterial and venous blood. The peroneal nerve presented a remarkable an-

giomatous appearance, as if threads of veins had been woven throughout the five or six inches which were exposed. In the absence of neuromata or any evidence of scarring

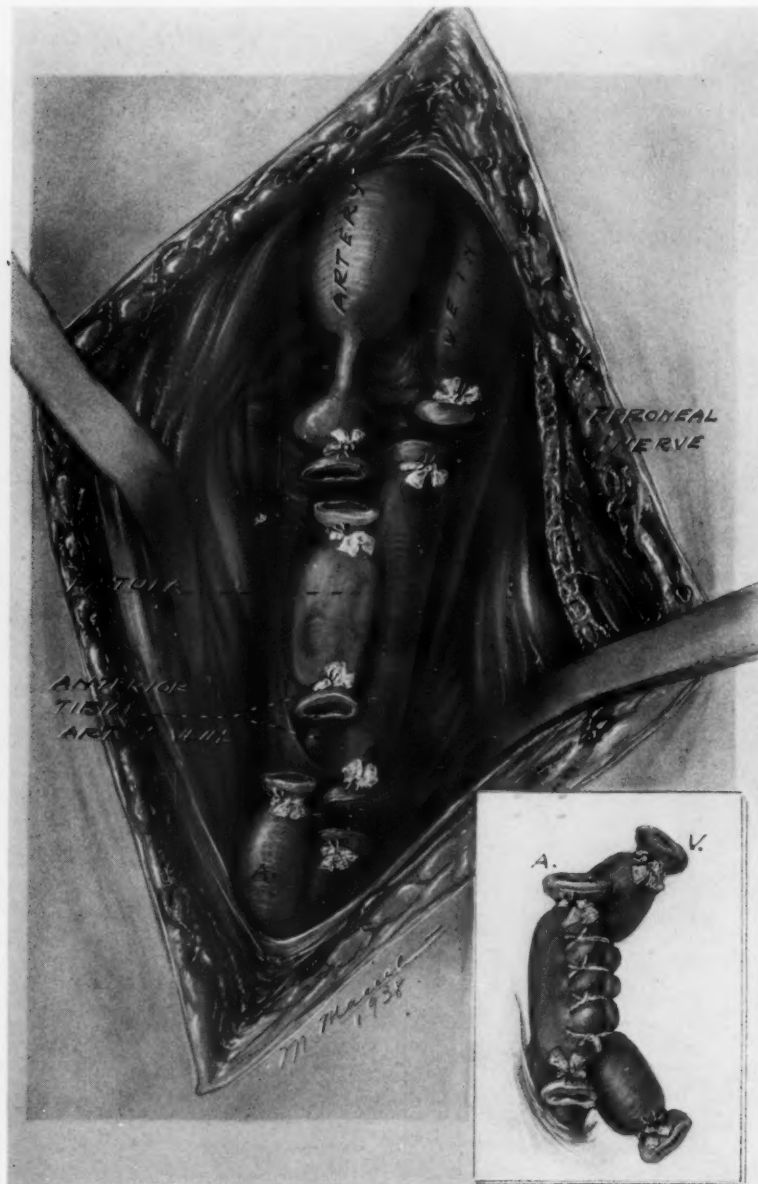


FIG. 2.—Case 13: The operation as completed. The quadruple ligations were effected with tape and anchored with transfixion sutures of silk. The fistula was closed with transfixion sutures (see insert) of braided silk because of the technical difficulty of exposing the anterior tibial vessels from a posterior approach.

or injury, we attributed its partial paralysis to this vascular change within it and, as we shall see from future observations, that was correct. The fistula was just at the bifurcation of the popliteal artery and vein—a very difficult situation because of the

fixation of all vessels at this point by the opening in the interosseous membrane. It was not particularly difficult to free the popliteal vessels down to the point of their bifurcation and the site of the arteriovenous aneurysm; with more difficulty the posterior tibial vessels beyond the communication were dissected free. The fistula being located practically in the foramen of the interosseous membrane, the freeing of the anterior tibial vessels from a posterior approach presented a vastly more difficult problem and would have resulted in prolonging the operation considerably. Finally it occurred to us to terminate the operation as illustrated—ligation and division of the popliteal vessels near the fistula; ligation of the posterior tibial vessels just beyond the fistula; and transfixing occluding sutures of the fistula just opposite the origin of the anterior tibial vessels. The fistula was not excised. At the conclusion of the operation no thrill could be felt; with a sterile stethoscope no bruit could be heard. Throughout the operation silk and tape, varying in size to correspond to the size of the vessels to be ligated, were employed. The wound was closed with fine silk, without drainage.

At the completion of the operation the circulation of the foot appeared better, the pedal pulses stronger, and the granulations of the ulcer redder and more healthy. The operative wound healed without any complications. Five days after the aneurysmal operation, the patient developed acute appendicitis and was operated upon for it. Except for this, his convalescence was exceedingly easy and he was discharged 17 days after admission.

Subsequent Course.—The circulation in his foot continued to improve and the ulcer promptly began to heal. Three months after the operation, it was completely healed. On November 22, 1932, Doctor Ober performed a "Z" plastic operation to lengthen the Achilles tendon. Within six months all evidence of any paralysis of the peroneal nerve had disappeared. At the present time (November, 1937) the patient has no disability or evidences of circulatory embarrassment in the right leg. There are no signs of an arteriovenous fistula.

In the interval between the first and second operations, the patient was confined to the use of crutches in order to get about; within a year after the second operation, he was again skiing and enjoying his usual sports without any apparent disability.

Case 24.—Cincinnati General Hospital No. 73329: A colored man, age 30, was admitted to the hospital April 16, 1937, with an enormously enlarged heart, auricular fibrillation and cardiac decompensation. He was dyspneic, the liver was enlarged and there was ascites. Râles were present at the bases of both lungs. A loud systolic murmur could be heard over the heart. The left leg was swollen and edematous. A large arteriovenous aneurysm was evident in the left groin at about the level of the profunda femoris artery (Fig. 6). The artery proximal to this point was enormously dilated. Pulse was very irregular, averaging about 79 at the wrist, but there was a marked pulse deficit. Blood pressure 160/50.

Fifteen years previously he had received a gunshot wound in the left groin, which was not followed by severe bleeding and did not require hospitalization. The patient performed hard manual work as a railroad fireman until four months before entering the hospital, although on careful questioning he had had some dyspnea for the previous two years. The patient dated his illness back only four months when he was forced to go to bed because of dyspnea, anginal attacks, and swelling of the abdomen. After a short rest he resumed work, but was soon forced to bed by another attack of cardiac decompensation and severe cardiac pain. This story repeated itself once or twice until, finally, after being in bed for ten days, he agreed to come to the hospital.

Two days after admission to the hospital he had a chill, his temperature rose to 101° F.; the left leg became markedly swollen. He evidently had developed extensive thrombophlebitis in the left leg. Another chill occurred three days later, the ascites increased; the patient became progressively worse in spite of all therapy which included, principally, morphine for his pain and digitalis for his cardiac failure. He became cyanotic and orthopneic; a severe cough developed.

On April 21, 1937, five days after admission, his venous pressure in the right arm was 25 cm. of water with no change after occlusion of the aneurysm. Three days after entering the hospital his blood pressure, with the fistula open, was 140/40; and with it closed 190/90. During this test it was noted that pulses became palpable in the foot when the fistula was occluded.



FIG. 3.—Case 24: Enormously dilated vessels exposed; revealing the region of fistula buried in dense scar tissue.

With the patient steadily going down hill, and developing a decubitus ulcer, it was felt that his only hope was an attempt to eliminate surgically the arteriovenous fistula.

Operation.—May 8, 1937: Under local anesthesia and with the patient in the sitting or orthopneic position, the operative procedure as illustrated in Figures 3, 4 and 5 was undertaken. The aneurysm was not excised, both because of the technical difficulties in

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dealing with the vein and the serious condition of the patient, but the fistula was firmly occluded by transfixion sutures of braided silk, after dividing the artery proximal and distal to the fistula and ligating the vein distally (Fig. 5).

The femoral artery was the largest artery Reid had ever ligated—more than one inch in diameter. Its wall was so thin that blood actually oozed through it proximally



FIG. 4.—Case 24: Femoral artery above and below fistula divided; vein distal to fistula ligated.

when the ligature of tape was tied. This was a precarious moment, but fortunately the bleeding stopped under gentle pressure with gauze.

Immediately after eliminating the fistula the patient volunteered the information that his heart had not felt "so good in 15 years." The thin-walled, oozing artery was reinforced by an overlapping with Scarpa's fascia. The wound was closed with silk, with-

out drainage. The wound healed per primam, without any complications. The patient's postoperative course was uneventful, except for some fever for five days due to his phlebitis. The action of his heart became regular on the operating table and remained so afterwards. Three hours after the operation his pulse was 40 per minute. After remaining at this slow rate for two hours, it gradually began to rise to between 60 and 70 where it remained except for occasional lowering to a rate of 50. His blood pressure reached a peak of 190/100, two hours after the operation, and gradually came down to 130/90, where it seemed to be stabilized at the time of the patient's discharge from the hospital, 23 days postoperative.

Subsequent Course.—This patient has been watched closely since leaving the hospital. The heart has decreased markedly in size, as shown by the teleoroentgenograms

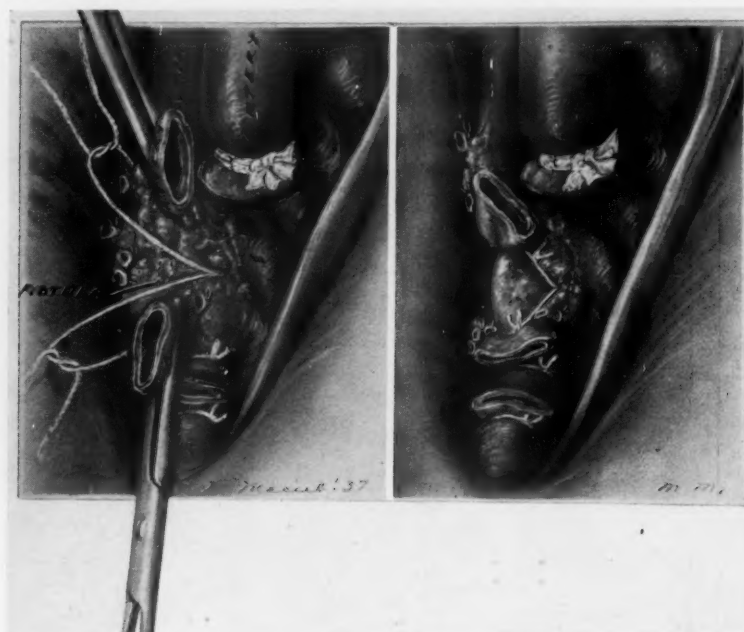


FIG. 5.—Case 24: Femoral vein opposite fistula pierced with two strands of braided silk which were divided to form a V and then tied so as to occlude the fistula completely.

(Fig. 7). The murmur has disappeared. The heart rate is regular at 80. Blood pressure 110/75. The venous pressure is 5 cm. of water. The swelling of the leg has disappeared. There are good pulses in the left foot. The ascites has disappeared. The liver has returned beneath the costal margin. The patient is able to work without any difficulty except for the annoyance of an elastic stocking which he wears for varicose veins of his left leg.

Case 18.—Holmes Hospital No. 340642: A white woman, age 35, was accidentally shot April 17, 1934. The bullet entered the left scapular region, passed through the axilla and lodged in the left anterior chest wall. Immediately after she was shot, she was unable to move the left hand and arm, which felt numb. At the time of admission to her local hospital the hand had become swollen, and she developed a large hematoma, about the size of a "football," in the region of the left axilla. She was hospitalized for two weeks, during which time the arm was abducted and elevated. There was a gradual decrease in the swelling of the hand, as well as that in the axilla. The nerve sensation improved except for the areas innervated by the ulnar nerve, which apparently remained completely paralyzed. Before leaving the hospital she was conscious of a noise which she could hear in

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her left ear. A loud bruit and thrill were demonstrable. She was told that she had an aneurysm.

She returned to the local hospital, May 18, 1934, and was operated upon, "at which time she lost a lot of blood." The bullet was said to be lodged in the vein at the site of the fistula. The vein was tied and the wound was packed with gauze. She remained in the hospital one month, and during this time there was considerable decrease in the swelling of

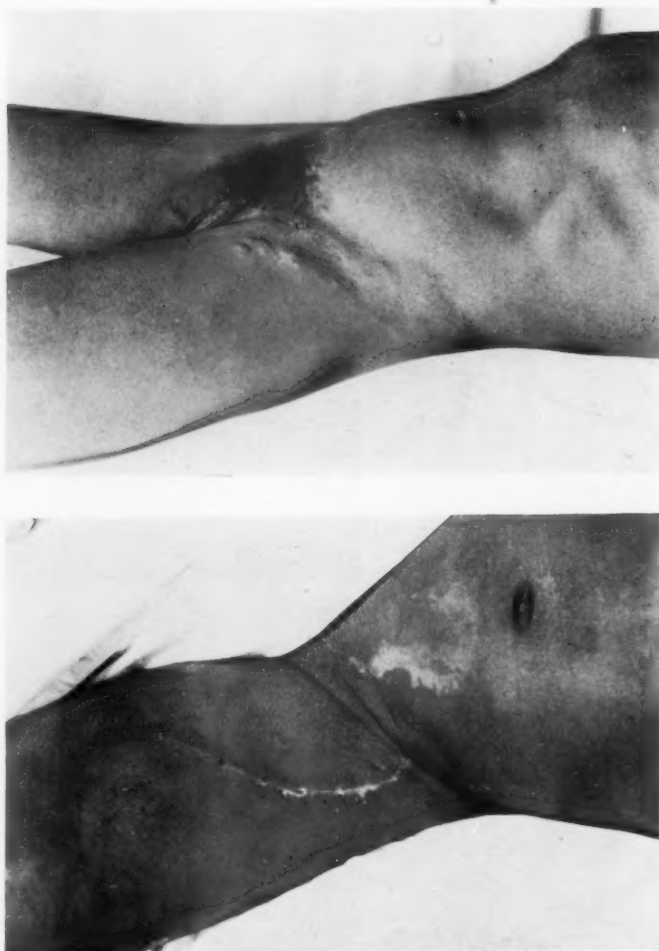


FIG. 6.—Case 24: Region of fistula before operation and ten days afterward. The curved incision was made in order to avoid the tremendously dilated veins.

the axilla and arm. About the middle of September, 1934, there was noted a return of the bruit and thrill. Also at this time there was a slight increase in the swelling of the arm; the veins about the shoulder became prominent. From that time on until her admission to the Holmes Hospital there was a gradual increase in the numbness of the left hand and, in addition to the paralysis of the ulnar nerve, she developed a typical wrist-drop, and practically a complete uselessness of the hand.

On admission to the Holmes Hospital, November 19, 1934, she presented a large swollen left arm, a big irregular scar which extended from just below the inner end of the clavicle to the humerus at the attachment of the pectoralis major muscle. About the



FIG. 7.—Case 24: Teleroentgenograms of the heart. No. 1: Before operation. No. 2: Three and a half months after operation. No. 3: Five months after operation. No. 4: Seven months after operation. No. 5: Ten months after operation.

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middle of this scar, and approximately one inch below the clavicle, there was an area of pulsation accompanied by a very pronounced thrill and bruit. This bruit could be heard

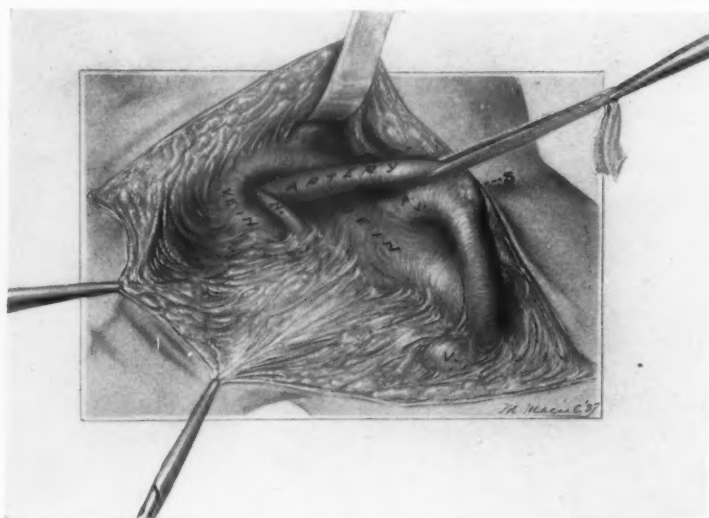


FIG. 8.—Case 18: Artery held up showing the superior arteriovenous fistula; scar (S) in artery of previous inferior fistula; nerves buried in scar tissue; and blind ends of inferior vena comites.

down the arm to the left radial artery. There were no cardiac murmurs. The blood pressure in the right arm was noted to be elevated ten mm. of Hg. when the fistula was oc-

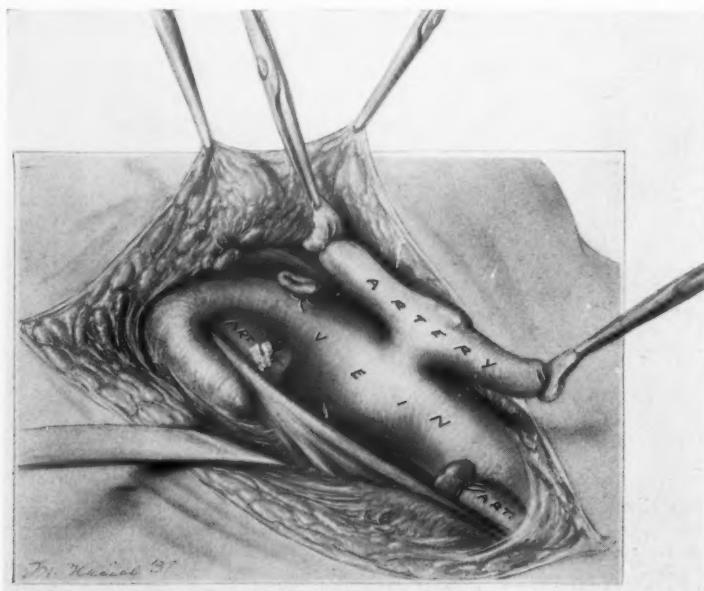


FIG. 9.—Case 18: Artery resected and held up; nerves partially freed. Note scar in artery from previous operation and blind ends of inferior vein.

cluded by pressure. The pulse dropped from 86 to 72 on one observation, and from 100 to 84 on another when the subclavian artery was occluded. The fingers of the left hand

were markedly atrophied. The thumb was involuntarily abducted. The patient was unable to flex or extend the wrist. There was a moderate loss of sensation over the anterior aspect of the lower third of the left forearm. The blood pressure in the left arm

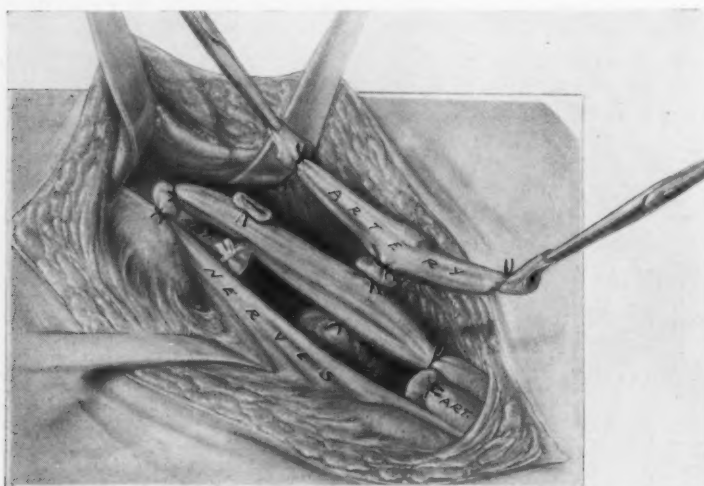


FIG. 10.—Case 18: Sites of ligatures in the process of excising artery and vein.

was 88/56; in the right arm with the aneurysm open, 102/66, and with the aneurysm closed, 112/76. Oscillometric readings in the left arm were a maximum of 3 units and in the right a maximum of 5. The measurements of the left arm, after the swelling had subsided, showed a decrease of about 1 cm., as compared with the right.

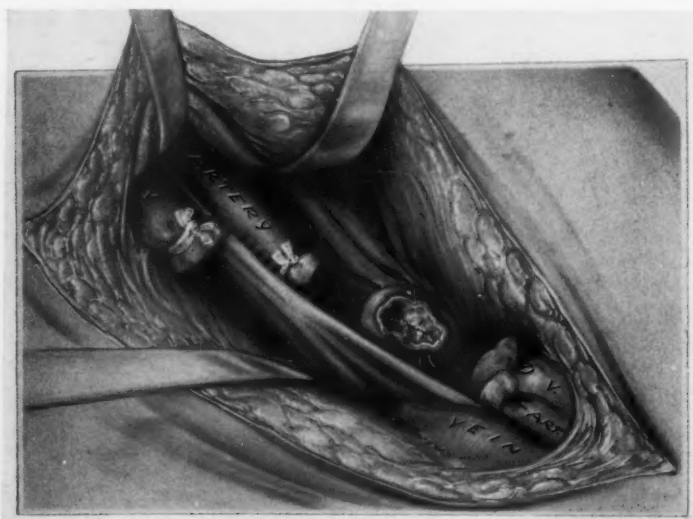


FIG. 11.—Case 18: Operation as completed. Axillary artery and vein excised; nerves freed.

A teleoroentgenogram of the heart showed no definite enlargement; and there was no change in its size when the fistula was closed. On fluoroscopic examination there was no change in the size of the heart, even though a slowing of 20 beats per minute occurred when the fistula was closed.

Second Operation.—Holmes Hospital, November 22, 1934: It required four hours

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of painstaking dissection to excise the scar tissue and to free the nerves, as well as to expose the site of the fistula. The operation is best appreciated by referring to Figures 8, 9, 10 and 11. A fistula was found between the axillary artery and a vein which lay above the artery. An old scar in the artery was also found, as well as the occluded vein, the result of the first operation. It was apparent that the bullet had pierced two veins and the artery, establishing a double arteriovenous fistula; that is, a communication between the artery and the vein above it and the vein below it. The first operation cured one of these fistulae, but had not affected the other.

After excising the scar tissue, which was exceedingly difficult to free from the nerves, the axillary artery and vein were removed. Silk was used for ligating the stumps of the vessels. The wound was closed without drainage. Following the operation there was no impairment of circulation to the hand; in fact, it was definitely improved, although

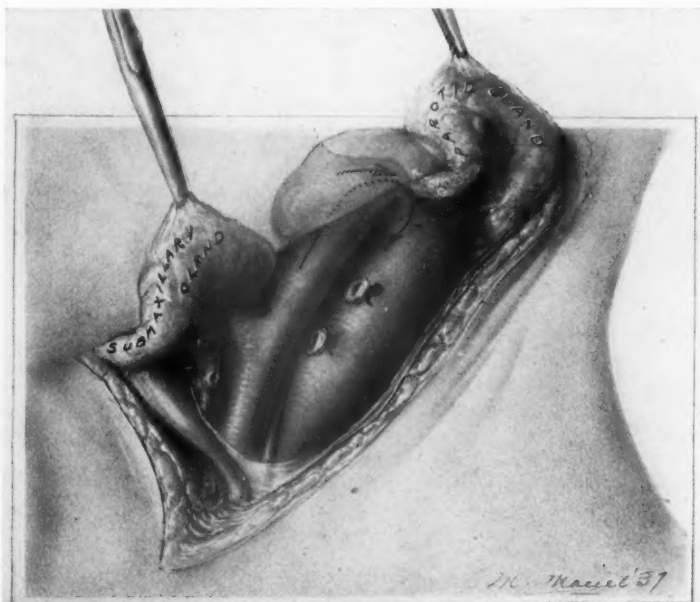


FIG. 12.—Case 23: Arteriovenous fistula at base of skull. Impossible to dissect jugular vein and internal carotid artery above it. False aneurysm in artery.

no radial pulse could be felt. The wound healed without any difficulty and the patient was discharged 13 days postoperative.

Subsequent Course.—This patient has been followed at various intervals since the operation. On March 23, 1937, there was no paralysis of the arm or any impairment of sensation. There was very slight weakness. All of the nerves seemed to have completely recovered their function.

The last examination was made in January, 1938, at which time there was no impairment of function or sensation in her arm or hand. The arm was not swollen. The interossei muscles of the hand had not returned completely to their normal size, although there was no disability in the use of these muscles.

Case 23.—Cincinnati General Hospital No. 63741: The patient, a colored man, age 53, was admitted first to the hospital August 12, 1933, suffering from multiple gunshot wounds of the left chest and one at the angle of the left jaw. Associated with this wound in the neck there was a large hematoma which in the course of about three days developed the typical characteristics of an arteriovenous aneurysm. A roentgenologic examination showed the bullet lying in the soft parts of the neck just to the left of the

midline. In its course it had penetrated the angle of the jaw but the fracture did not extend entirely through the mandible. The size of the heart was carefully noted by means of a teleroentgenogram. The gunshot wounds healed without complications and the patient was discharged September 30, 1933. It was our plan to postpone the operation for two reasons: First, to allow an adequate time for the development of collateral circulation; and second, to see if the fistula would close spontaneously. The patient was

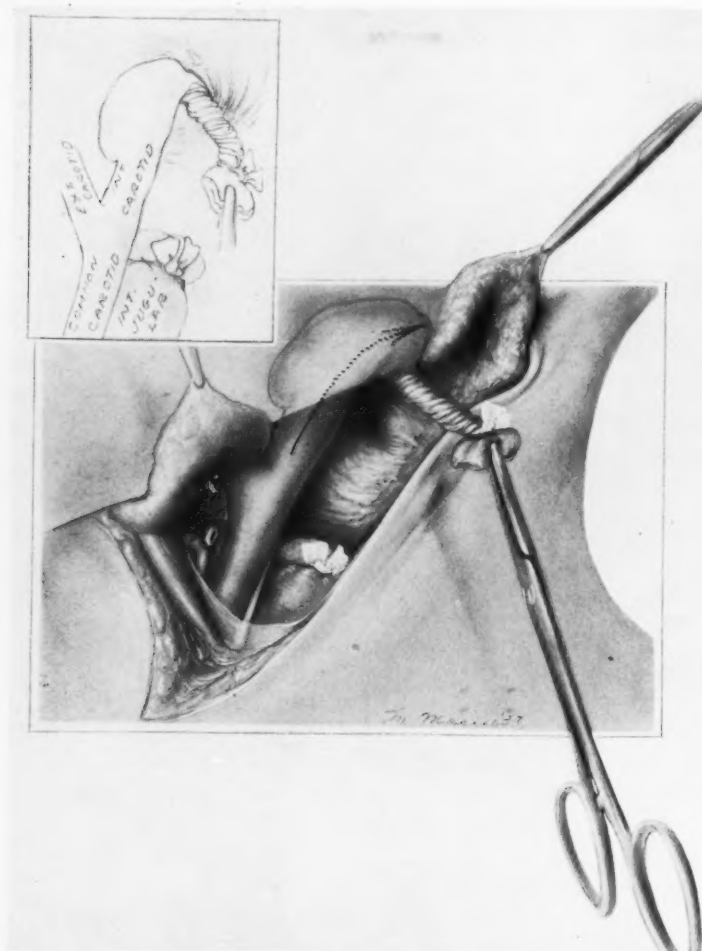


FIG. 13.—Case 23: Jugular vein divided and being twisted up so as to occlude arteriovenous fistula.

watched in the Out-Patient Clinic from the time of his discharge until his readmission, October 29, 1936. He consented to come in at this time for operation because of increasing pulsation in his neck, ringing in the ear and spells of dizziness. During the two months preceding this admission, he had fallen backwards and to the left during these attacks of dizziness. In addition to these symptoms he had had some questionable difficulty in speaking and intermittent blurring of vision, associated with diplopia and an increased sweating of the left face.

At this time there was noted a swelling between the angle of the jaw and the lobe of the ear which was about the size of an egg. This was definitely pulsatile and ex-

hibited a very loud thrill and bruit. The bruit could be heard up as far as the temporal region and down the neck to the clavicle. There were no enlarged veins in the subcutaneous tissue of the neck or face. No weakness could be detected in the eye muscles. The fundi appeared to be normal. The patient complained of having double vision when he tried to look straight forward; in any of the lateral, superior or inferior directions he did not see double. There was no demonstrable weakness in the extremities of either side. Oscillometric readings were normal in all extremities. Venous pressure in the arm was 62 Mm. of water.

An electrocardiogram showed sinus tachycardia T_1 was diphasic and of low voltage. *Diagnosis:* "Minimal evidence or myocardial disease." A roentgenogram of the skull, on this last admission, showed several metallic fragments in the neck, one of which was at

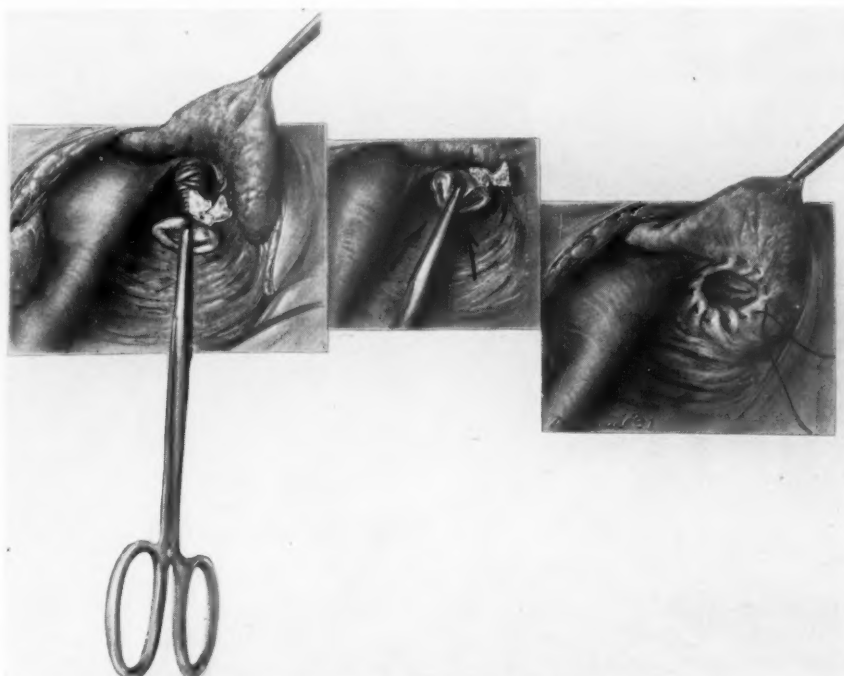


FIG. 14.—Case 23: Jugular vein twisted, pushed into jugular foramen and anchored so as to occlude the arteriovenous fistula.

the level of the second cervical vertebra. The bones of the skull were apparently normal. No demonstrable enlargement of the heart could be noted. Branham's bradycardic phenomenon was not demonstrable. Blood pressure in both arms before the operation was 124/86.

Operation.—November 6, 1934: Under local anesthesia, the carotid artery, its branches, the jugular vein and vagus nerve were carefully freed up to the level of the posterior belly of the digastric muscle. This dissection was rather tedious because there were some large branches of the jugular vein which had to be carefully dissected free, ligated and divided. The fistula was located just at the base of the skull, and in the artery opposite the fistula there was an aneurysmal dilatation about the size of an olive. This extended up to the foramen in the skull. Communication with the internal jugular vein was so near to the base of the skull that one could not possibly free the vein beyond this point. The internal carotid artery was quite tortuous and about twice its normal size. When the dissection was fairly well completed, it was possible to see the arterial



FIG. 15.—Case 23: Photograph of specimen removed at autopsy three years and eight months after operation. Fistula closed; arterial aneurysm healed.

blood squirting into the jugular vein, forming whorls which were easily visible through the wall of the vein.

The operator was never in quite such a quandary; it was obvious that the involved artery and vein could not be excised because it would be impossible to ligate the distal stumps of the excised vessels. For some time the internal carotid artery and jugular vein were compressed at the same time; this, of course, appeared to stop the thrill and bruit, but the patient after several minutes began to become dizzy and showed obvious cerebral anemia. This convinced the operator that it would be necessary to try, at all hazards, to preserve the continuity of the internal carotid artery. The fistulous connection was so high that it could not be isolated enough to ligate it, neither could sutures be placed directly through the wall of the vein at this point. Finally, the jugular vein was divided low in the neck and the distal portion twisted until it definitely occluded the arteriovenous communication (Figs. 12, 13 and 14). After this, the twisted jugular vein was pushed forcibly

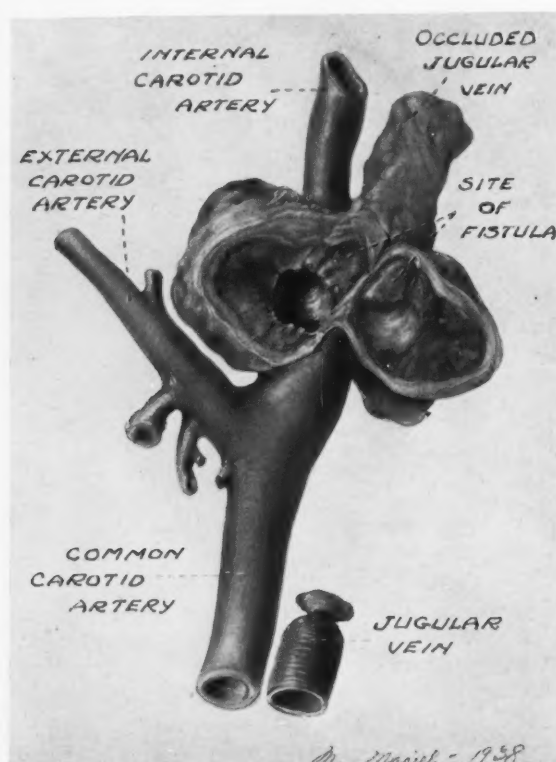


FIG. 16.—Case 23: Illustration of specimen removed at autopsy, with flap of healed arterial aneurysm turned back, showing site of fistula and restored lumen of the internal carotid artery.

up toward the jugular foramen of the skull and held in this position by means of anchoring sutures, and a purse string suture, which caught some of the parotid gland and the fascia

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in the bed of the jugular vein. This kept the twisted segment of vein from untwisting and kept it in position to block the communication. At the completion of the operation, and before closure of the wound, no bruit could be detected by means of a sterilized stethoscope. The wound was closed with interrupted, fine silk sutures.

It was interesting to note the fluctuations of the blood pressure during the course of the operation. The operation was begun at 8:40. About one-half hour later, when the carotid sinus was being carefully dissected, there was a noticeable rise in the blood pressure, reaching a peak of 160/110. The pressure when the operation was begun was 120/100. The elevated blood pressure was maintained for about one and one-half hours and then gradually dropped down to a little bit below what it had been during the manipulation. It was a very good clinical illustration of the rise in blood pressure during the manipulation of the carotid sinus. The operation required about three hours. At 1:00 P.M., about one hour after the operation, the blood pressure had gone back up to 170/110. It was still at this level at ten o'clock on the evening of the operation. By 9:30 o'clock of the next morning the blood pressure had dropped to 85/60; at 11:30 it was 94/64. On November 8, it was 120/80. On the ninth, it was 135/80, and on the succeeding days, up to the time of the patient's discharge from the hospital, November 18, 1936, it ranged around 135/95.

Follow-Up.—When this patient was seen in the Follow-Up Clinic, January 20, 1937, there was no evidence of any arteriovenous communication. The patient's symptoms had practically disappeared. His only discomfort was a slight annoyance due to the division of the twelfth nerve at the time of operation. It was necessary to sacrifice this nerve in order to free the vessels sufficiently far up toward the communication.

The patient was sent for, February 1, 1938, for a follow-up study, and it was found that he was suffering from extensive bilateral pulmonary tuberculosis. He was admitted to the hospital and died from his tuberculosis about two weeks later. The autopsy showed that the fistula was completely healed and that the false aneurysm of the artery was filled with rather firmly organized blood clot. Through this clot the artery had assumed more or less its normal size. The vein distal to the fistula was also occluded (Figs. 15 and 16).

Case 29.—Cincinnati General Hospital No. 80815: The patient, white, male, age 18, was admitted to the hospital October 10, 1937, with a gunshot wound of the right popliteal space. The bullet entered approximately the middle of this space and came out in the anterior part of the lower leg at about the junction of its upper and middle thirds. There was a considerable hematoma, but no serious external bleeding. Within 24 hours the presence of an arteriovenous aneurysm was evident from the characteristic thrill and bruit. The patient was kept in the hospital for 18 days, during which time the wound healed without infection; careful observations were made upon the heart and peripheral circulation. At the time of his discharge no pulse could be felt in the right foot, but the circulation seemed to be adequate. No nerve injuries were noted on admission, nor had any developed before he left the hospital. He was sent home to wait for the development of an adequate collateral circulation and the absorption of the hematoma before considering any curative surgical procedures.

He was seen again in the Follow-Up Clinic December 19, 1937, with no special complaints except a tendency of the right leg to become cold. At that time no pulses could be felt in the right foot. The hematoma had become largely absorbed and the tissues of the popliteal space were becoming soft. The signs of the fistula were more evident and there were no evidences of cardiac disability.



FIG. 15.—Case 23: Photograph of specimen removed at autopsy three years and eight months after operation. Fistula closed; arterial aneurysm healed.

blood squirting into the jugular vein, forming whorls which were easily visible through the wall of the vein.

The operator was never in quite such a quandary; it was obvious that the involved artery and vein could not be excised because it would be impossible to ligate the distal stumps of the excised vessels. For some time the internal carotid artery and jugular vein were compressed at the same time; this, of course, appeared to stop the thrill and bruit, but the patient after several minutes began to become dizzy and showed obvious cerebral anemia. This convinced the operator that it would be necessary to try, at all hazards, to preserve the continuity of the internal carotid artery. The fistulous connection was so high that it could not be isolated enough to ligate it, neither could sutures be placed directly through the wall of the vein at this point. Finally, the jugular vein was divided low in the neck and the distal portion twisted until it definitely occluded the arteriovenous communication (Figs. 12, 13 and 14). After this, the twisted jugular vein was pushed forcibly

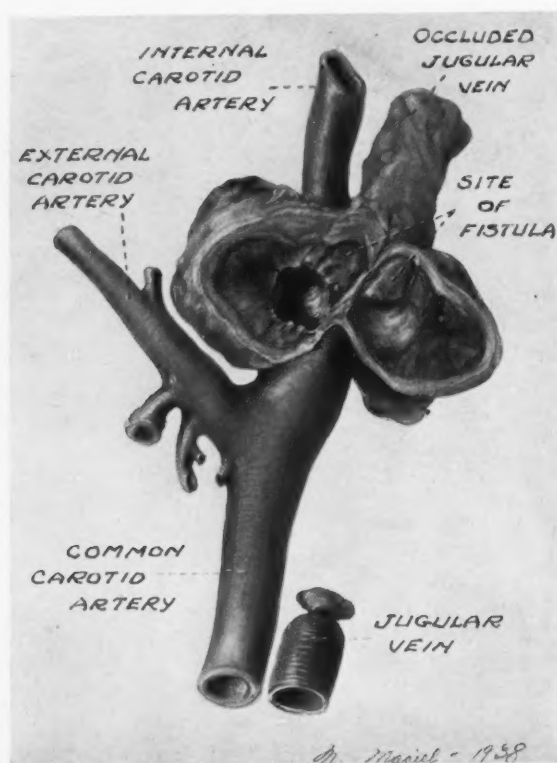


FIG. 16.—Case 23: Illustration of specimen removed at autopsy, with flap of healed arterial aneurysm turned back, showing site of fistula and restored lumen of the internal carotid artery.

up toward the jugular foramen of the skull and held in this position by means of anchoring sutures, and a purse string suture, which caught some of the parotid gland and the fascia

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in the bed of the jugular vein. This kept the twisted segment of vein from untwisting and kept it in position to block the communication. At the completion of the operation, and before closure of the wound, no bruit could be detected by means of a sterilized stethoscope. The wound was closed with interrupted, fine silk sutures.

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The patient was sent for, February 1, 1938, for a follow-up study, and it was found that he was suffering from extensive bilateral pulmonary tuberculosis. He was admitted to the hospital and died from his tuberculosis about two weeks later. The autopsy showed that the fistula was completely healed and that the false aneurysm of the artery was filled with rather firmly organized blood clot. Through this clot the artery had assumed more or less its normal size. The vein distal to the fistula was also occluded (Figs. 15 and 16).

Case 29.—Cincinnati General Hospital No. 80815: The patient, white, male, age 18, was admitted to the hospital October 10, 1937, with a gunshot wound of the right popliteal space. The bullet entered approximately the middle of this space and came out in the anterior part of the lower leg at about the junction of its upper and middle thirds. There was a considerable hematoma, but no serious external bleeding. Within 24 hours the presence of an arteriovenous aneurysm was evident from the characteristic thrill and bruit. The patient was kept in the hospital for 18 days, during which time the wound healed without infection; careful observations were made upon the heart and peripheral circulation. At the time of his discharge no pulse could be felt in the right foot, but the circulation seemed to be adequate. No nerve injuries were noted on admission, nor had any developed before he left the hospital. He was sent home to wait for the development of an adequate collateral circulation and the absorption of the hematoma before considering any curative surgical procedures.

He was seen again in the Follow-Up Clinic December 19, 1937, with no special complaints except a tendency of the right leg to become cold. At that time no pulses could be felt in the right foot. The hematoma had become largely absorbed and the tissues of the popliteal space were becoming soft. The signs of the fistula were more evident and there were no evidences of cardiac disability.

He was seen again January 23, 1938, when practically the same observations were made. At this time he complained of a little pain in the right knee joint after using his leg for a considerable time.

By February 10, 1938, it was felt that there was sufficient collateral circulation to undertake a curative operation. Besides, the studies of the leg showed that the proximal artery was definitely dilated. This was confirmed both by palpation and oscillometric studies. Average pulse, 65; average blood pressure, 100/64. No pulse could be felt below the knee. On numerous observations the patient exhibited the typical Branham's bradycardic phenomenon, the average drop in pulse rate on closure of the fistula being nine beats. On a few observations there were no striking changes in the blood pressure



FIG. 17.—Case 29:—Popliteal arteriovenous aneurysm. Large false arterial aneurysm opposite fistula and extending anteriorly. Communication is with only one branch of the popliteal vein, which divided high.



FIG. 18.—Case 29: Complete extirpation of the involved popliteal vessels.

after occluding the fistula. There were no noticeable changes in the general venous pressure following occlusion of the fistula. In the left femoral vein it was $4\frac{3}{4}$ cm. of water when the fistula was open and the same when it was occluded. In the right femoral vein it was $5\frac{3}{4}$ cm. of water with the fistula open and $4\frac{1}{2}$ cm. when it was occluded. In the right and left antecubital vein the venous pressure remained about 10 cm. of water with the fistula open or closed. The circulation time in the right femoral vein averaged, with the fistula open, 13.6 seconds; with it closed, 17.3 seconds. The circulation time in the left femoral vein, with the fistula closed, was 14.6 seconds and 15.1 seconds with it open. The circulation time as measured in the left antecubital vein was 16.8 seconds when the fistula was open. Roentgenologic studies did not demonstrate any appreciable enlargement of the heart. On fluoroscopy, the pulmonary conus was moderately prominent with unusually vigorous pulsations. The heart became approximately one-quarter smaller with occlusion. The blood volume was 5,200 cc. October 28, 1937, and on February 12, 1938, it was 4,900 cc.

Operation.—February 16, 1938: Ether anesthesia. The patient was placed on his abdomen and put in the Trendelenburg position so that the right leg would be well above the level of his heart. The operation consisted of excision of the femoral artery and vein. The fistula was located about one-half inch above the bifurcation of the femoral artery. At this point the femoral vein was already divided into two vena comites. The fistula communicated with only one of these. In the artery opposite the fistula there was a false arteriovenous aneurysm about the size of an English walnut. This projected directly anteriorly. The operative procedure is illustrated in Figures 17 and 18. The operation was rendered rather difficult because of the danger of injuring the nerves; also, because of the fact that the vessels could not be very well delivered, as they were tied down by the anterior tibial branches of the femoral artery. The large vessels were ligated with double braided silk, the smaller vessels with very fine silk. The proximal artery was definitely dilated to about half again its normal size.

The knee was completely immobilized by a crinoline encasement. On the following morning good pulses could be easily detected in both the dorsalis pedis and posterior tibial vessels. These have remained good, although the volume has lessened somewhat since about the third day postoperative.

Three weeks after operation the pulse was 48; blood pressure, 102/60; and blood volume, 5,300 cc. In the right and left arms and right femoral vein, the venous pressure was 7.75 cm. of H₂O. The circulation time in the right femoral vein was 17.6 seconds. In the right arm the circulation time was 19.6 seconds.

Case 25.—Cincinnati General Hospital No. 73338: The patient, white, male, age 19, was admitted to the hospital April 16, 1937, because of a pulsating mass below and behind the right ear. This was not causing any trouble, except that he wished to have it removed in order to be made eligible for admission into the U. S. Navy. He had previously been in the hospital in September, 1936, for the repair of a left, indirect inguinal hernia. It was not noted at that time that he had a cirroid aneurysm of his neck. The lesion had evidently grown considerably between that admission and the one of April, 1937. The patient believed that there had been something wrong with the blood vessels in his neck since infancy. There was no history of any injury. It in no way incapacitated him, but he was becoming more and more conscious of a roaring noise in the right ear, especially when lying down. He had engaged in strenuous work and athletics without any evidences of cardiac disability.

Just behind and below the right ear there was a visible pulsation, over which one could feel a strong thrill and on auscultation hear a loud and typical to-and-fro arteriovenous bruit. The carotid artery on this side appeared to be definitely enlarged. Compression of it caused the bruit to disappear and the swelling to collapse. The external jugular vein was much enlarged and carried a strong bruit. Firm pressure directly below the tip of the ear made this bruit almost disappear. When the patient bent his head forward the swelling became definitely larger and he occasionally felt a slight numb sensation in the region of the ear. However, none of the nerves were apparently involved.

A teleroentgenogram of the heart showed no enlargement. Blood pressure in the right arm averaged 120/60, and in the left arm, 100/50. Neither blood pressure was noticeably affected by occluding the fistula. The pulse was 70 and it was not slowed by pressure over the aneurysm. Venous pressures in the right arm were 12.5 cm. of water, and in the left, 11 cm. Circulation time in the left arm was 16.4 seconds. Blood volume, 6,950 cc.

Operation.—May 4, 1937 (Figs. 19, 20, 21 and 22): Local anesthesia. In the process of dissection the digastric and stylohyoid muscles, the posterior occipital, external, internal and the common carotid vessels, the hypoglossal nerve and the lower margin of the parotid gland were all exposed. Occlusion of the external jugular vein at a point low in the neck caused rapid dilatation distal to the occlusion, and through the wall of the

vein arterial blood could be seen swirling from the force of the arterial communications. Occlusion of the external carotid artery stopped the swirling, the bruit and the thrill.

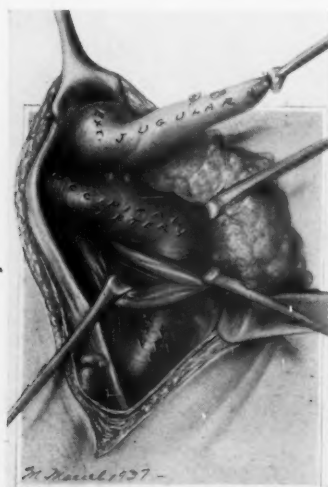


FIG. 19.—Case 25: External jugular vein divided and held up, showing communications with the huge occipital artery. External carotid is also very large.

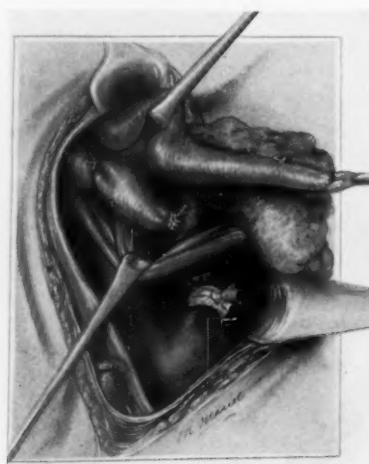


FIG. 20.—Case 25: Two ligatures on the occipital artery; one on the external carotid. Ligation of three definite arterio-venous communications.

Occlusion of the internal carotid artery did not affect it. The external carotid artery was noticeably dilated, measuring 1.4 cm. in diameter, while the internal carotid artery measured only 0.8 cm. in diameter. The posterior occipital artery was tremendously enlarged,



FIG. 21.—Case 25: Twisting of the external jugular vein to occlude any other fistula which may be higher.

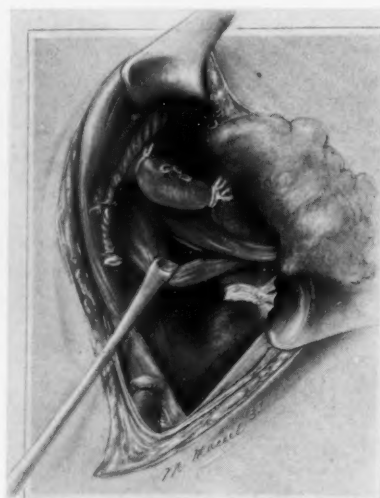


FIG. 22.—Case 25: Twisted external jugular vein anchored to the sternomastoid muscle, in order to prevent its untwisting.

measuring 0.9 cm. in diameter. During the process of freeing the bifurcation of the common carotid artery, the pulse dropped from 100 to 80.

The occipital artery was ligated twice with braided silk: first, at its point of disappearance behind the parotid gland and again at the point of its origin from the carotid

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artery. The external carotid artery was ligated with heavy tape just above its origin from the common carotid.

The external jugular vein was then freed low in the neck and divided. It was dissected upward to the point of its disappearance deep in the parotid gland. In this region numerous vessels communicating with the occipital artery were encountered. As these communicating vessels were ligated, activity within the vein became less, but not until one high up in the neck, approximately one-quarter inch in diameter, was ligated did the vein collapse and all activity stop. At this time, no bruit could be heard through a sterile stethoscope. The patient said that the noises in his head, to which he had become accustomed, had ceased.

In order to obliterate any other communications which might not have been discovered, the long segment of external jugular vein was twisted until it became a very small cord. This was anchored to the sternomastoid muscle in such a way that it could not untwist. Closure was then made with interrupted, fine silk sutures. Following the operation there was no noticeable change in the pulse rate. This patient has been seen on numerous occasions since the operation, the last time being February 5, 1938, at which time there was no evidence of any recurrence of the cirroid aneurysm.

Immediately after the operation, the blood pressure in the left arm was 140/25; this was at 1:00 P.M. By 11:00 P.M. the blood pressure was 120/60. On the following day, it was 110/65, and thereafter it ranged around 115/60. This fluctuation in blood pressure immediately after operation was no doubt connected with manipulation of the carotid sinus. On February 9, 1938, the venous pressure was 11.5 cm. of water in the right arm. Circulation time in the left arm was 12.4 seconds; in the right arm, 12 seconds. Blood volume on May 15, 1937, was 5,670 cc., and on February 9, 1938, was 6,430 cc.

Case 28.—Cincinnati General Hospital No. 81335: The patient, white, male, age 16, was admitted to the hospital October 22, 1937. On October 3, 1937, a pistol shell exploded and a small fragment of it hit the inner side of his right upper arm about three inches above the elbow. He bled profusely, losing about a pint of blood, until he received first aid. On the day following this accident, the upper arm became markedly swollen; this continued for three days and then gradually began to subside. With the subsidence of this swelling a small pulsating tumor appeared at the site of the injury, over which a thrill and bruit were noted. The swelling continued to increase rapidly and the skin over it became very thin; he also developed a weakness in the gripping of his hand, and a tingling sensation and an inability to use his hand well. The pain at the site of the injury became increasingly severe.

Physical Examination.—At the time of admission to the hospital he had an extensive, though not complete, paralysis of the median nerve and severe causalgia in the forearm and hand. The swelling above the elbow (Fig. 23) measured about 4 cm. in



FIG. 23.—Case 28: Arteriovenous aneurysm of brachial vessels. Skin near rupturing. Rapidly progressing paralysis of median nerve. Operation 23 days after injury by a piece of steel.

diameter. The skin at the site of the puncture wound was very thin and there was real danger of spontaneous rupture. Over this swelling a thrill and bruit, characteristic of arteriovenous aneurysm, were very pronounced. Radial pulse could be felt, though markedly lessened. The blood pressures were the same—120/60 in both arms. Pulse

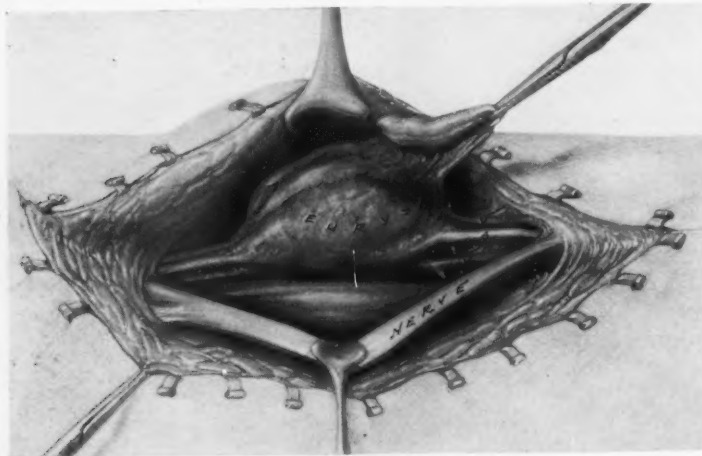


FIG. 24.—Case 28: Nerve retracted from its bed in aneurysm, where pressure was causing paralysis. Some very thin skin being excised with the aneurysm. Skin is protected by towels clipped to its edges.

80, and did not slow down on occluding the aneurysm, but the diastolic pressure rose 30 Mm. Hg. Venous pressure before operation was 9 cm. of water in the left arm and 10½ cm. of water in the right arm. Circulation time, as determined by the NaCN method, was 21.30 seconds in the left arm, and 14.0 seconds in the right arm, proximal

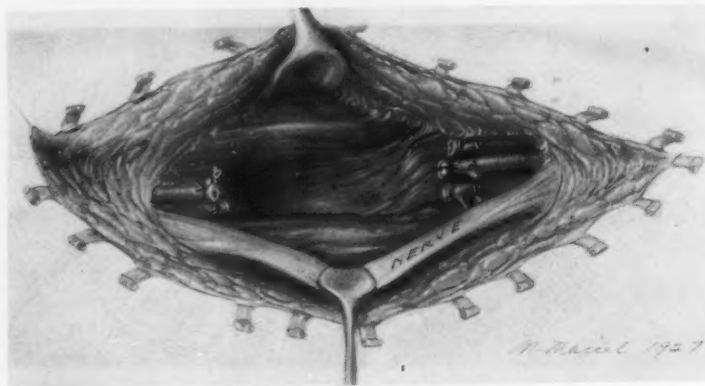


FIG. 25.—Case 28: Aneurysm completely excised. Vessels ligated with silk.

to the fistula. The heart was normal in size. An electrocardiogram was normal. Due to the progressive median nerve palsy, the severe causalgia and the danger of rupture of the aneurysm, immediate operation was decided upon.

Operation.—October 26, 1937 (Figs. 24, 25 and 26): Both of the vena comites communicated with the divided artery. Both the proximal and distal ends of all vessels involved were patent. The aneurysm was completely excised. The median nerve was stretched over the aneurysmal sac and definitely flattened. It had not been injured by the fragment of shell, but was obviously being damaged by the pressure and pulsations of

the aneurysm. Following operation there were no complications and no appreciable embarrassment to the circulation of his hand and forearm. The pulse and temperature remained normal. He was discharged November 4, 1937. Blood pressure 100/70; venous pressure left arm, 8 cm. of water, circulation time left arm, 20 seconds.

Follow-Up.—January 9, 1938: Sensation and muscle power had all returned to normal. A faint radial pulse could be felt.

Case 27.—Christ Hospital No. 112543: The patient, white, male, age 33, height 6 feet, weight 156 pounds, was admitted to the hospital, September 8, 1937, with the diagnosis of an aneurysm of left arm. He had consulted a physician because of nervous spells, vague gastro-intestinal complaints, and shortness of breath on exertion. These symptoms had gradually increased during the past six months. There was found on further questioning that in association with the shortness of breath, there was palpitation, but no precordial pain and no edema of ankles. Examination revealed an arteriovenous fistula of the left arm which it was thought explained many of the symptoms. The fistula had evidently developed 16 years before, at the age of 17, at which time he sustained a compound fracture of the left humerus just above the elbow. This was reduced and placed in a plaster encasement for several days, after which a secondary reduction was undertaken. When the encasement was removed some time later, he was told he had an aneurysm, which was subsequently operated upon. The patient does not recall whether his arm was swollen, what the condition of the circulation in the fingers was or when the operation was performed. He does, however, remember being told that at the operation he had nearly died from loss of blood, and that it would be dangerous for him to be operated upon again, and that if he were careful, he would not have any trouble. He therefore refrained from athletic sports and never considered having the fistula operated upon.

Physical Examination.—The general examination was essentially negative. The patient was tall and thin, and somewhat nervous. There was no evidence of cardiac decompensation. There was some deformity of the left arm due to the old fracture, with some loss of the carrying angle. There was a large, broad scar on the cubital region extending up the inside of the arm. Underneath this scar was a visible, pulsating mass with the typical thrill and bruit of an arteriovenous fistula. The brachial artery entering it was quite large and felt about 1 cm. in diameter. There was no dilatation of the peripheral veins, leading to the surmise that the vein distal to the fistula had been ligated, a fact subsequently confirmed at operation. There was a very weak pulsation felt in the left radial artery, and the circulation in the fingers was good. Obliteration of the fistula by pressure directly over it caused the pulse to drop from 88 to 60 within one minute. Percussion of the heart indicated that it was moderately enlarged, but the teleoroentgenogram did not reveal any enlargement. Without occlusion of the aneurysm, the blood pressure in the right arm was 100/70; with occlusion of the aneurysm it was 125/85. Blood pressure in the left arm was 130 systolic; murmur was not heard until 110, maximal at 95. Circulation time, right antecubital vein, with 0.35 cc. NaCN, was 15 seconds without occlusion. Venous pressure right antecubital vein without occlusion of fistula was 8.5 cm. H₂O; with occlusion, 8.5 cm. H₂O. Blood volume 5,400 cc., 8.17

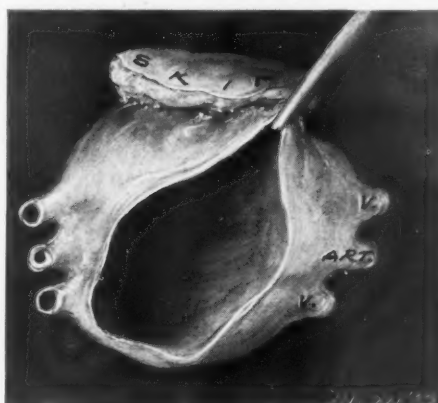


FIG. 26.—Case 28: Window cut in the side of aneurysmal sac. Both veins and the artery were divided, and communicated from within the false aneurysmal sac.

per cent body weight. Hematocrit, 43.2 per cent. Plasma volume, 3,060 cc., 4.53 per cent body weight. The electrocardiogram was normal.

Operation.—September 11, 1937: The brachial artery was about 1 cm. in diameter proximal to the fistula. The vein, proximal to it, about 1.5 cm. in diameter. The



FIG. 27.—Case 27: Arteriovenous aneurysm of brachial vessels; duration, 16 years. Vein, distal to fistula, ligated over 15 years ago.

vein, distal to the fistula, was a thin fibrous cord, and had apparently been ligated previously. The artery, distal to the fistula, was about 7 to 8 Mm. in diameter. Occlusion of the artery, proximal to the fistula, reduced the blood flow through the fistula approximately 60 per cent, but occlusion of the distal artery was necessary to stop the flow. The artery, proximal and distal to the fistula, was ligated with one-half inch tape; the

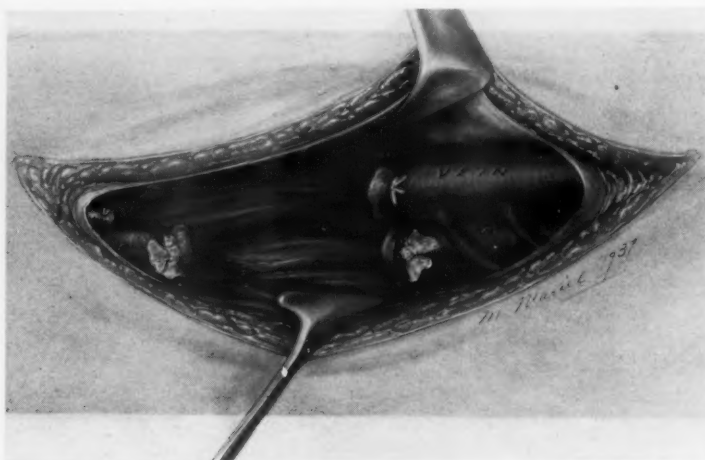


FIG. 28.—Case 27: Operation as completed.

vein, proximal, with braided silk; the small vein, distal, with medium silk; the fistula-bearing vessels were excised (Figs. 27 and 28). There was a good pulse at the left wrist immediately after operation.

Subsequent Course.—Thirteen days postoperative: Blood pressure, right arm, was 115/75; pulse, 80; circulation time, 19.5 seconds; venous pressure, 10 cm. of water; blood volume, 4,800 cc., 7.2 per cent body weight; hematocrit, 42.25 per cent; plasma

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volume, 2,760 cc., 4 per cent body weight. On September 30, 1937, the teleoroentgenogram showed no change. On December 20, 1937, the heart had become slightly smaller, and all of the patient's former complaints had disappeared. The brachial artery had become much decreased in size.

Case 21.—Cincinnati General Hospital No. 56203: The patient was admitted to the hospital March 9, 1936, with a left subclavian arteriovenous aneurysm. He had previously been in this hospital, in December, 1933, with advanced tuberculosis of both apices. The aneurysm had resulted from a gunshot wound with a 22 caliber bullet, March 22, 1935. Following this injury there was mild shock but very little bleeding. Shortly after, the patient noticed that his left arm was partially paralyzed and that he had a peculiar noise in the region of the left shoulder. He said that it sounded like the exhaust of steam. The motion and sensation in the left arm gradually improved up to the time the patient was first sent into the hospital for careful studies. Blood pressure, 126/60; venous pressure, in the right arm, 5.2 cm. water; while in the left arm, the venous pressure was 9 cm. water. Circulation time in the left arm, 15 seconds. A teleoroentgenogram showed a slight dilatation of the aorta. The paralysis in his arm involved primarily the ulnar nerve, but this was not complete. Due to the patient's extensive pulmonary tuberculosis, the positive Wassermann, and the absence of any serious cardiac damage, it was decided to postpone any operative procedure. The patient was discharged after five days' study and told to rest as much as possible because of his tuberculosis.

He was readmitted, May 13, 1936, for a period of 12 days for further study. For the past year he had been taking much rest at home. At this time it was noted that all signs of a communication between the artery and vein had disappeared. There was a faint systolic bruit, probably due to the variation in the size of the artery, but no evidence whatever of a characteristic to-and-fro arteriovenous murmur. Apparently the fistula had closed spontaneously, as a result of the patient's rest in bed. On several occasions subsequently, this patient has been seen, but showed no evidence of an arteriovenous fistula.

Case 4.—Children's Hospital No. 2356: The patient, white, male, age 8, was admitted to the Children's Hospital, March 12, 1928, with an eye condition for which he had previously consulted Dr. D. T. Vail, Jr. In June, 1927, eight months previously, the child had injured his left eye by running a small piece of chicken wire into the eyeball. This puncture was apparently into the lower inner quadrant of the eye, although no scar of it could be detected. Six weeks later the eye became reddish and he was treated for pink-eye. By September, 1927, the left eyelids, particularly the upper lid, were noted to be swollen, and the conjunctival veins had become very large and very red. Later, the supra-orbital vein became large, and varicosities appeared in the eyelids and on the nose around the inner canthus of the eye. When he was first seen by Doctor Vail, a few days before our operation, an aneurysm of the eye was detected. The conjunctival veins were very large and distended with arterial blood. They, as well as the larger varicosities of the eyelids, nose and forehead, definitely pulsated. There was no edema, which is usually very marked when there is an intracranial arteriovenous fistula between the cavernous sinus and internal carotid artery. The retinal veins were distended and pulsated. Vision did not appear to be disturbed. On light palpation over the closed eye, a faint thrill could be felt. A definite arteriovenous bruit could be heard over the eye, nose and forehead. Temporary occlusion of the common carotid artery caused the eye to recede, the veins to collapse partially and to cease pulsating, and the thrill and bruit to disappear. Not knowing exactly where the perforation had occurred, and because the most marked signs of the cirroid aneurysm were at the inner angle of the eye, it was thought that the abnormal communications were probably between branches of the external carotid artery and the neighboring veins.

Operation.—March 13, 1928: The bifurcation of the common carotid artery was first

exposed. Here we were surprised to find that temporary occlusion of the external carotid artery had no effect on the aneurysm but that occlusion of the internal carotid completely stilled it. Consequently, the external carotid artery was permanently ligated with a heavy silk ligature, and the common carotid was occluded by a removable aluminum band. Through a Killian incision over the left tear sac and extending into the brow, the supra-orbital and other large veins in the exposed area were excised. Another small incision in the upper eyelid allowed the removal of a large vein. All ligations were made with silk ligatures.

Subsequent Course.—For five days after the operation, the patient vomited very frequently. On the third day a slight weakness of the right face and of the right arm was noted. This paralysis did not progress, and disappeared entirely in about two weeks. We interpreted the vomiting, as well as the paralysis, as being due to cerebral anemia. During this period of uneasiness, it was a great comfort to know that we could remove the metallic band if the symptoms demanded it.

September 27, 1930: Examination showed that the dilated conjunctival veins had almost completely disappeared. There was no exophthalmos. No enlarged veins in the eyelids, about the nose or on the forehead were seen. Auscultation over and about the eye revealed no bruit. There was no evidence of any right-sided weakness. Mental development appeared to be normal.

In the neck, the left common carotid artery was about one-quarter as large as the right, and definitely pulsated. At the site of the occluding band, a loud, harsh systolic bruit could be heard such as one can hear when an artery is markedly constricted. Four months after the original operation, the artery was apparently totally occluded, as neither a murmur nor a pulsation could be detected. Since that time, and now, there has been a partial restoration of the lumen with a return of function in the artery.

April 13, 1938: Both eyes appeared to be normal. Vision and muscular action were undisturbed. Retinal vessels did not pulsate and have returned to normal size. The fistula remained closed. No thrill or bruit was demonstrable either over or about the eye.

The band could not definitely be felt. The left carotid artery was of normal size and compression of it caused a good temporal pulse to disappear. Compression of the right common carotid artery did not affect the left temporal pulse. The artery, just distal to the band, was not demonstrably enlarged. A soft systolic bruit, indicating only a mild constriction, could be heard over the site of the band. Evidently the lumen of the artery at the site of the band has been restored almost to normal size.

Case 30.—Cincinnati General Hospital No. 88134. The patient, white, male, age 19, was admitted to the hospital, February 9, 1938. He had four cirroid aneurysms (Fig. 29)—one in the neck, two in the left forearm and one on the dorsum of the left foot. All of them appeared spontaneously. The two on his left forearm appeared about ten or 12 years ago without any antecedent injury, so far as he knew. They increased slowly in size, except during the past year or two, when he did not note any particular change. Three weeks before admission the smaller of these two lesions on the left forearm, the one situated just above the wrist, was excised by Dr. George Curtis of Columbus, Ohio. He reported the lesion removed as being an arteriovenous aneurysm.

The next lesion to appear was in the left side of his neck. The patient first observed this about three or four years ago. It grew rapidly and recently interfered somewhat with his voice, which frequently became "husky."

The lesion on the dorsum of the left foot was noted about the same time as the lesion in his neck. However, it had not grown nearly so rapidly.

None of these lesions ever caused the patient any pain. He continued to work hard and did not note any increase in shortness of breath or forcible heart action. There was never any swelling of his ankles. The patient at no time was conscious of any noise connected with the tumors.

Physical Examination.—The patient's face was slightly congested, which condition

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the patient himself had been conscious of. In the left side of his neck, extending across the midline to the right side, there was a very large pulsating mass, which presented irregularities due to obvious large blood vessels beneath the skin. The skin was not telangiectatic and appeared to be normal. It moved freely over the lesion. The size and extent of this lesion are well depicted in Figure 29.

On palpation, there was a very pronounced thrill over the entire surface of the swelling, most marked over the lateral margin of the sternomastoid muscle. All of the vessels pulsated very forcibly, especially an extremely large one just below the left submaxillary gland. The bruit was very loud, particularly at the point where the thrill was most easily felt. This bruit was transmitted up into both temporal vessels. Curiously enough, it was a little more pronounced on listening over the right temporal



FIG. 29.—Case 30: Patient with four spontaneous cirroid aneurysms. A very large one in the neck, a small one in the left foot, and two in the left forearm (lower tumor of arm removed, elsewhere, three weeks before). All lesions, especially that in the neck, pulsated vigorously, and exhibited the typical thrills and bruits of arteriovenous aneurysms.

artery. The lesion did not seem to extend below the level of the clavicle or far up under the jaws. On compressing the carotid artery on the left side the thrill could be made to disappear, but there remained a faint bruit. On compressing both carotid arteries the bruit became almost inaudible. One could not detect any definite increase in the size of either carotid artery. When the patient bent forward, his face became rather quickly and markedly flushed, almost cyanotic.

On the volar surface of the left forearm there was another pulsating tumor, the center of which was situated about 8 cm. below the internal condyle of the humerus. The size of this tumor was roughly that of a large English walnut. Its vertical diameter was 5.5 cm.; the transverse diameter about 5 cm. This tumor pulsated and presented an irregular contour, due obviously to blood vessels beneath the surface of the skin. The skin, however, was normal and moved freely over the swelling. The color of this lesion was slightly bluish, due unquestionably to the blood in the vessels beneath

the skin. A faint thrill could be felt over this tumor; there was a very definite and typical arteriovenous bruit. The bruit and thrill could be made to disappear by occluding the brachial artery, which appeared, on palpation, to be about normal in size.

Farther down in the forearm there was a recent wound which measured about 7 cm. This was the site of the operation three weeks ago. The radial and ulnar vessels appeared to be normal. A tumor, quite similar to the one described in the forearm, only about one-quarter the size, was situated on the dorsum of the foot. The skin over it was normal. It was not quite so irregular, but a characteristic arteriovenous bruit was audible over the mass. The pulsation and the bruit disappeared on compressing the femoral artery. The femoral artery appeared to be normal. This patient had definite cardiac enlargement and a slight systolic murmur at the apex.

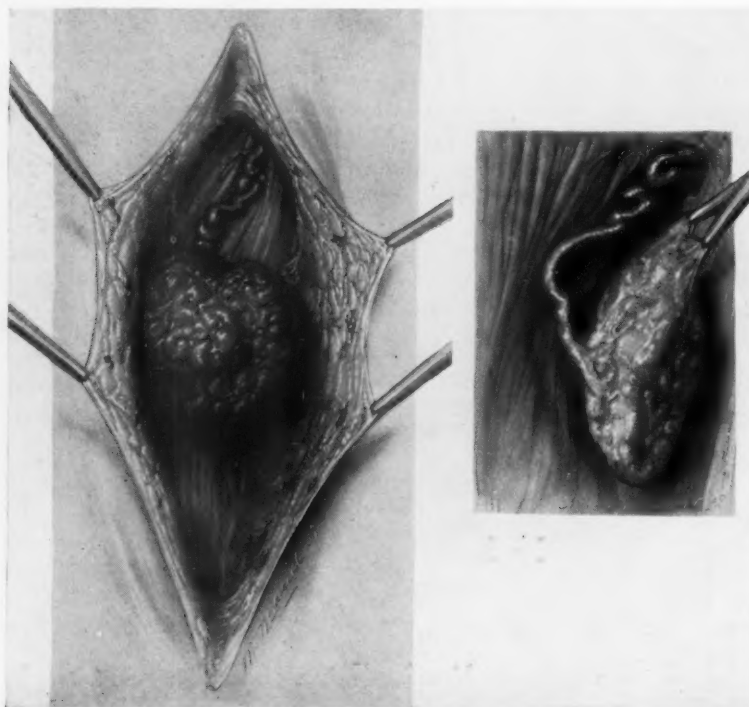


FIG. 30.—Case 30: Dissection of the aneurysm from left forearm, except for one large, tortuous anastomosing artery. Before dividing this vessel the aneurysm pulsated and exhibited the signs of a cirroid.

Closure of the large communications in the neck by direct pressure caused a definite slowing in the heart rate of 20 beats, and the blood pressure in the right arm changed from 130/65 to 140/78.

First Operation.—February 12, 1938: The cirroid aneurysms of the left arm and on the dorsum of his left foot were removed under local anesthesia. In the case of the arm it was easy to demonstrate two or three large arteries running directly into, and communicating with, the mass of blood vessels. It was possible to dissect the entire mass free except for the largest of these communicating arteries. The mass would still pulsate when the artery was open, but would cease on occluding it. Following the removal of the tumor, injection of this main artery with Hill's solution filled all the blood vessels in the mass (Fig. 30).

In the case of the foot there were two arteries close to the dorsalis pedis artery

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which ran directly into the cirroid aneurysm (Fig. 31). There was no direct communication with the dorsalis pedis. Closure of these two communicating arteries stopped the activity of the aneurysm. The excision of this tumor was relatively easy compared with that in the arm. In both instances the ligations were made with silk and the wounds were closed with interrupted silk sutures without drainage.

Following these two operations there was no noticeable change in the cardiovascular mechanism of this patient (Fig. 32). The wounds healed without any difficulty.

Second Operation.—February 26, 1938 (Figs. 33, 34, 35, and 36): Ether anesthesia. An attempt was made to do something to the huge cirroid aneurysm which involved both sides of the neck, especially the left. An incision was made along the border of the sternomastoid muscle from the tip of the ear almost to the clavicle and then curved in front of the throat across the midline. In the subcutaneous tissues countless small blood vessels were encountered and progress was necessarily very slow. We were finally able, however, to expose the carotid artery and jugular vein from the clavicle up above the bifurcation of the common carotid artery. The jugular vein was about three times its normal size. Through its wall could be seen whirling arterial blood, and there was an actual tremor, which presented the thrill and the bruit which were so pronounced before operation. Low in the neck there were two large branches which connected the jugular vein with the huge vessels making up the cirroid aneurysm. When these were divided the jugular vein immediately collapsed to about its normal size,



FIG. 31.—Case 30: Aneurysm of foot, showing two distinct communicating vessels. No direct connection with dorsalis pedis artery.

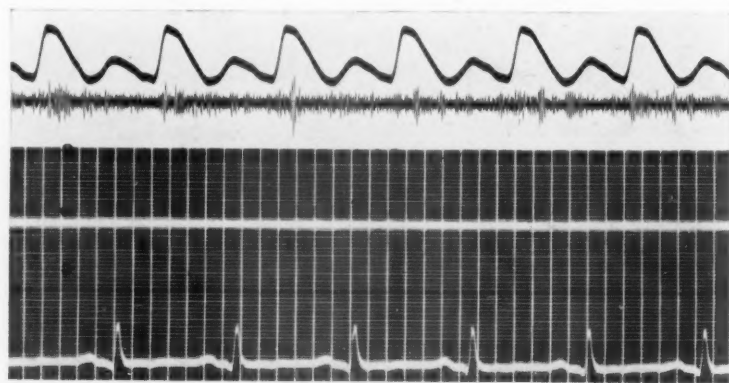


FIG. 32.—Case 30: Simultaneous electrocardiogram; radial pulse tracing; and sound tracing showing continuous murmur over carotid artery.

the thrill and bruit disappeared from it and it was possible to detect a faint bruit only over the rest of the cirroid aneurysm, through a sterile stethoscope. Following this procedure the bifurcation of the common carotid artery was carefully freed and, first, the superior thyroid artery was ligated. This reduced the bruit, but did not make it disappear

entirely. The external carotid and ascending pharyngeal arteries were then ligated, following which the huge cirroid aneurysm almost ceased to pulsate.

The next procedure was to locate and ligate the inferior thyroid artery. After doing this, the flap of skin and subcutaneous tissue were dissected off the surface of the aneurysm to the midline of the neck and up to a point above the hyoid bone. Starting laterally an effort was made to remove most of this mass of blood vessels. There was still some pulsation and the control of hemorrhage was a ticklish proposition. It was soon possible to determine that the lower two-thirds of the thyroid gland was not involved



FIG. 33.—Case 30: A, B, Two huge vessels from the cirroid aneurysm, emptying arterial blood into the jugular vein.

in the cirroid condition. The upper one-third of the thyroid was markedly involved, and the condition extended behind the trachea on to the esophagus and far over to the right side of the neck. The aneurysm extended also above the hyoid bone. The dissection was continued until it was obvious that no more could be removed from this side without the danger of injuring the esophagus. At this point, large transfixion sutures of braided silk were used to ligate the mass of vessels which had been largely freed. On doing this, a rather large hole was torn in a big blood vessel just behind the trachea, at about the level of the upper third of the thyroid gland. This was controlled by direct finger pressure until the operator secured a piece of muscle and plugged the hole by suturing it directly into the opening. It was noted at this time that the bleeding was still very active. After the hemorrhage had been controlled, the wound was closed with interrupted fine silk sutures, without drainage. A plaster encasement was applied to the patient's neck and head to immobilize the wound. Ligation of the right external carotid

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artery was considered, but this was deferred until a later date because the operation had already consumed about four hours.

Subsequent Course.—Following this operation there was a little difficulty in swallowing and talking for two days, but otherwise the convalescence was quite uneventful. The wound healed per primam. The left vocal cord was not paralyzed. The blood pressure in both arms on admission and for eight days after the neck operation ranged around 155/80; on leaving the hospital it was 124/88. The patient was discharged, March 27, 1938, with a definite bruit still audible over the front of the neck, but no thrill or pulsa-



FIG. 34.—Case 30: A, B. Large connecting veins, from cirroid to jugular vein, divided. Superior thyroid, external carotid and ascending pharyngeal arteries divided.

tions could be felt. This bruit could be made inaudible by compression of the right common carotid artery at its bifurcation but not by compression lower down in the neck. Evidently the reversed circulation through the circle of Willis was enough to keep the cirroid aneurysm active when only the common carotid artery was occluded.

Special studies of the heart in the Cardiac Laboratory showed: February 11, 1938: Circulation time right arm, 18 seconds; left arm, distal to the fistula, 21 seconds; left arm, proximal to the fistula, 11.6 seconds. Venous pressure, right arm, +8 cm. of water; left arm, distal to the fistula, +12 cm., proximal, +7.5 cm. Blood volume, 5,440 cc. March 21, 1938: Circulation time in left arm was 12.2 seconds. Venous pressure right arm, 5 cm. of water, left arm 5 cm. Blood volume, 5,070 cc.

Third Operation.—April 26, 1938: Under local anesthesia, the right external carotid artery was ligated. This operation, together with the previous one, effected a complete sacrifice of both external carotid arteries; there still remains, however, a faint audible bruit.

Case 16.—Holmes Hospital No. 340334: An instance of a large arteriovenous hemangioma involving the right forearm and hand. The patient, white, female, age 24, was admitted to the hospital June 18, 1934. The condition she presented was obviously congenital. It was not, however, noted by the parents until she was about three years old, when there was observed some irregularity and a slight enlargement on the volar surface of the right forearm. The condition gave the patient no trouble until about one year before admission, although the swelling had gradually increased in size. During the year previous to admission, the swelling seemed to have increased rather rapidly and

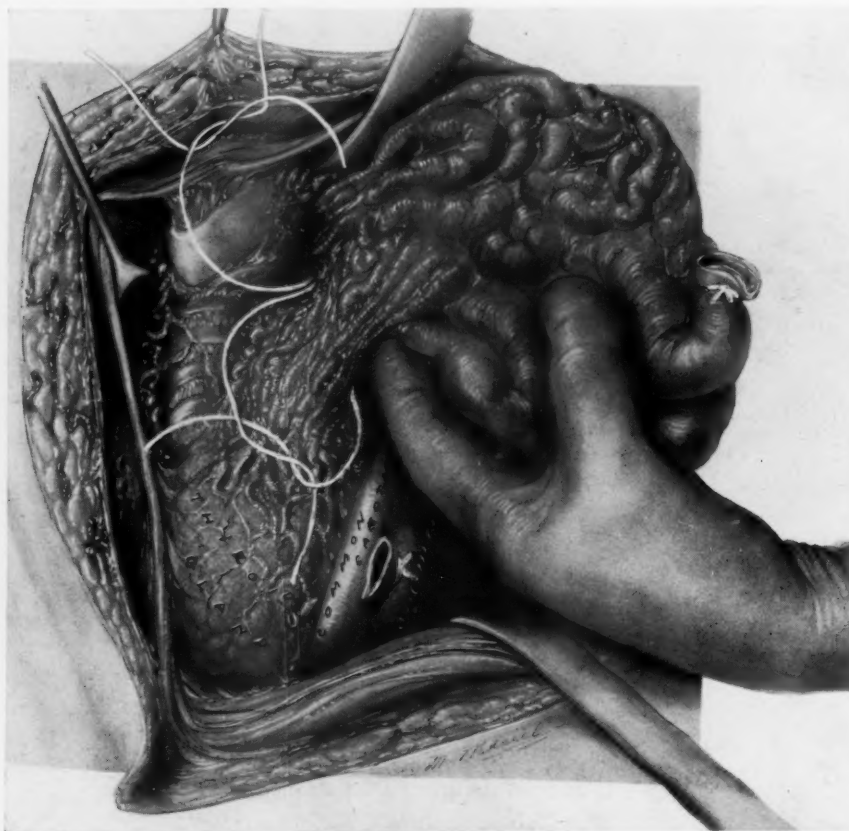


FIG. 35.—Case 30: Ligatures placed, preparatory to amputating a large portion of the cirroid aneurysm. Note the involvement of the upper third of thyroid gland, and the extension of the cirroid aneurysm behind the trachea into the right side of neck. Inferior thyroid artery is ligated.

had begun to cause her considerable trouble, particularly a sensation of discomfort and tightness when the arm was held down, and a rather excessive fatigability of the fingers, with a definite impairment of their function.

Physical Examination revealed an apparently healthy girl, except for the congenital vascular lesion involving the volar surface of the right forearm and hand (Fig. 37). The swelling was rather irregular and increased perceptibly when the arm was held down. There was definite diminution in the size when the arm was elevated. If the middle of the forearm was squeezed when the arm was hanging down, a definite fluctuation wave could be felt in the palm of the hand, accompanied by an increase of the swelling in the palm. In places there were light-bluish discolorations of the skin, but there was no definite involvement of the skin at any place by an angiomatous condition. The super-

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facial veins were quite large. No thrill and no bruit could be demonstrated. The radial and ulnar vessels were easily palpable. Throughout the extent of this vascular lesion one could feel small, hard bodies, which were thought to be calcified phleboliths (Fig. 38). The patient's blood pressure was essentially the same in both arms, namely, 105/78.

The patient had had so much trouble during the previous year that she had frequently been advised to have her arm amputated lest the growth would extend, rupture and cause fatal hemorrhage. With considerable hesitation, an operation to excise the lesion was undertaken.



FIG. 36.—Case 30: Operation as completed. Esophagus is definitely involved. Lesion extends behind trachea to the right side of neck. Faint bruit still audible through a sterile stethoscope.

Operation.—June 19, 1934: The incision extended from 4 cm. below the internal condyle of the humerus well into the palm of the hand at the base of the fingers. The operation was performed without a tourniquet, and with the arm elevated far above the level of the heart. The angiomatous condition involved the surfaces of practically all the muscles, the nerves and the space between the flexor profundus muscle and the interosseus membrane. The blood encountered, though it was not excessive, was definitely arterial. Due to the greatly elevated position of the arm, we were able to shave off from the muscles and the nerves the greater part of this lesion without necessitating the

tying of many blood vessels. The belly of the flexor sublimis digitorum to the index finger was so involved that it was thought best to excise it. The tendon of this muscle was later sutured to the tendon of the flexor sublimis digitorum of the third finger. The



FIG. 37.—Case 16: Photograph of cirroid aneurysm of right arm and hand before operation.

ligatures used were fine, black silk; the skin incision was closed with interrupted, fine, black silk sutures. The arm was placed in a plaster encasement and suspended as far as possible above the patient by means of a Balkan frame. Although it was impossible



FIG. 38.—Case 16: Roentgenograms of right arm before and after operation.

to remove all of this growth, it was felt that considerable thrombosis would occur in the remaining portion.

Subsequent Course.—The patient has been observed regularly since the operation, the



FIG. 39.—Case 16: Photographs one year after operation.

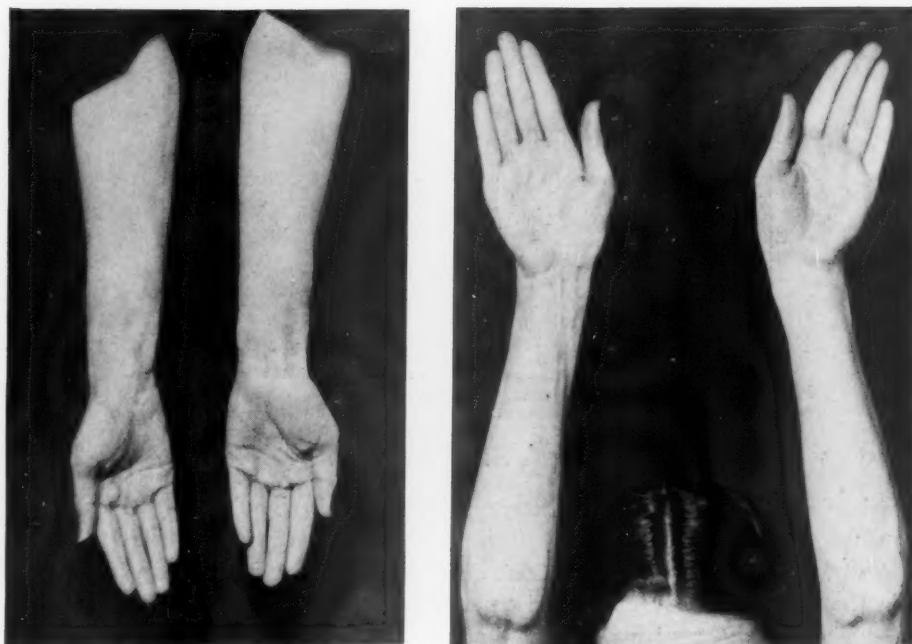


FIG. 40.—Case 16: Photographs three years after operation. Very slight recurrence of cirroid aneurysm. Function of arm and hand perfect.

last time being January, 1938, at which time there was practically no disability. The index finger apparently functioned quite normally (Figs. 39 and 40). There was a little swelling above the wrist and in the palm but this had not increased materially during the past year. It was noted, in June, 1936, that the muscular development of the arm had practically returned to normal.

Experimental Studies.—In connection with the study of our clinical cases there have naturally arisen many questions which we wished to study in the

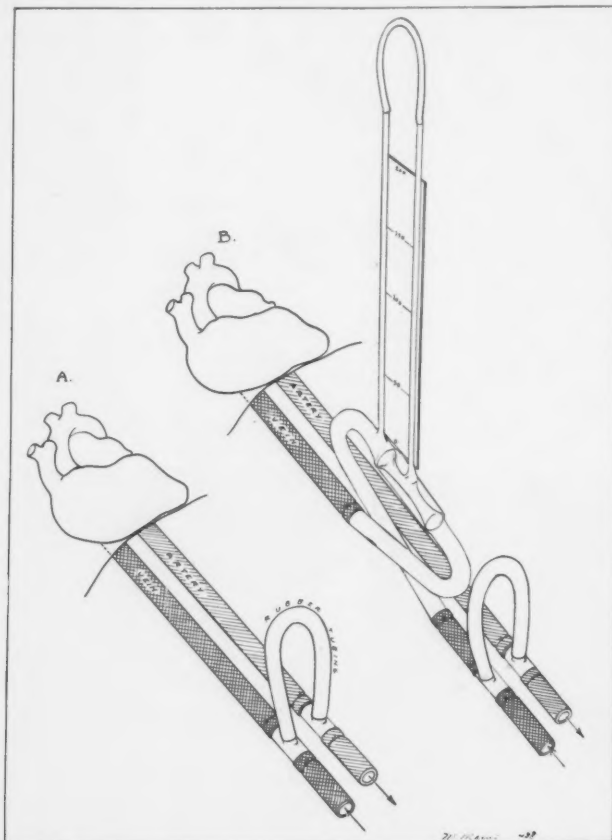


FIG. 41.—(A) Method of establishing an easily controllable fistula between the aorta and vena cava, used in some of our acute experiments. With the rubber tubing the fistula can be closed or opened as desired. Also, all the blood from the aorta can be easily diverted back into the heart. (B) Same as A; with a Venturi meter in the vena cava, to measure the volume of blood flow.

Laboratory of Experimental Surgery. We shall here refer only to experimental observations which we believe may shed some light upon our clinical studies or the previous observations of others who have interested themselves in the subject of abnormal arteriovenous communications.

The experiments have been both acute and chronic. In the acute, or non-survival experiments, communications were established between the abdominal aorta and vena cava (Fig. 41 A) in four heparinized dogs. In two of these animals, in addition to the abnormal arteriovenous communication, a Venturi meter (Fig. 41 B) was inserted into the vein between the site of the fistula

TABLE I
SYNOPSIS OF DATA RELEVANT TO TWENTY-ONE CASES OF ARTERIOVENOUS AND NINE OF CIRSOID ANEURYSMS
(1925-1938)

Case	Date; Hospital; Number	Age, Sex, Color, Occupation	Location of Lesion	Etiology	Duration	Type	Principal Symptoms and Signs	Operator, Date	Treatment	Result	Comments
1	1/1926 P. U. M. C. China.	23 yrs. Male. Chinese. Soldier.	Left femoral region.	Gunshot wound.	10 das.	Arteriovenous fistula between femoral artery and vein.	Thrill and bruit; infection; secondary hemorrhage.	Reid. Jan. 1926	Excision of fistula with quadruple ligation; also ligation of profunda artery.	Well.	Operation was forced by the infection and several secondary hemorrhages. Fistula at the level of the profunda artery. All vessels ligated with braided silk. No attempt to close wound or ends of ligated vessels. Thorough dakinization. Quick healing without the sloughing out of ligatures.
2	9/12/27 C. G. H. No. M-8420	29 yrs. Male. Colored. Laborer.	Cavernous sinus.	Blow with brass knuckles behind right ear.	4 wks.	Intracranial arteriovenous fistula of internal carotid artery and cavernous sinus.	Complete ophthalmoplegia; extreme exophthalmos; edematous and everted eyelids; poor vision; bruit over head and neck.	Reid. 1. 9/13/27 2. 8 hrs. later. 3. 9/27/27	1. Ligation of right external carotid artery and occlusion of common carotid artery by metallic band. 2. Removal of band from common carotid artery. 3. Second occlusion of common carotid artery with band.	Aneurysm well.	Case reported in detail in Am. Jour. of Surg., Oct. 1931. The first band was removed because of hemiplegia which promptly disappeared after its removal. Following the second band, there was temporary paralysis which cleared except for the left arm, which was spastic.
3	3/5/28 C. G. H. No. N-2403	35 yrs. Female. Colored. Laundress.	Right arm.	Stab wound.	9 yrs.	Arteriovenous fistula of right brachial artery and vein.	No disturbance from aneurysm. Thrill and bruit.	No operation.	None.	Unknown.	Patient entered the hospital for acute cholecystitis, which subsided. The aneurysm was discovered on physical examination and patient refused to permit operation upon it. The heart was slightly enlarged.
4*	3/12/28 Children's Hospital. No. 2356	8 yrs. Male. White. Child.	Left eyeball.	Perforation of eye with a piece of chicken wire.	9 mos.	Arteriovenous aneurysm of ophthalmic artery and vein.	Swelling of eyelids and forehead and marked congestion of conjunctival blood vessels; thrill and bruit.	Reid. 3/13/28	Ligation of external carotid artery; occlusion of internal with metallic band; ligation and excision of supra-orbital vessels.	Well.	For a few days after operation this patient had a partial weakness of the right side of the body, but it was not necessary to remove the metallic band. The fistula has remained closed and the sight of the eye good.
5	4/10/28 C. G. H. No. R-1054	29 yrs. Female. White. Secretary.	Right foot and ankle.	Fall of bench on dorsum of foot at age 7.	22 yrs.	Cirsoid aneurysm.	Extensive angiomatous condition of foot and ankle; thrills and bruit; bleeding from dorsum of foot; increase in size of foot.	Reid. 1. 4/16/28 2. 4/28/28 3. 5/12/28 4. 2/28/29 5. 4/1/29 6. 1/27/32 7. 2/5/32 8. 10/20/32 9. 11/21/32	1. Ligation of anterior tibial artery. 2. Ligation of dorsalis pedis artery. 3. Ligation of posterior tibial artery. 4. Ligation of large artery behind external malleolus. 5. Ligation of posterior tibial artery higher in leg. 6. Ligation of anterior tibial artery just below the knee. 7. Ligation of arteries about the ankle. 8. Excision of angioma of dorsum of foot; full-thickness graft. 9. A few pinch grafts applied to foot.	Improved.	In October, 1931 this case was reported in full in the Am. Jr. of Surg. Since then several operations have been performed, making a total of nine; the last one in 1932. The patient now has practically no disability but a faint bruit can be heard on listening over the ankle. Very few large superficial vessels.
6	7/12/28 St. Francis Hospital. Los Angeles.	57 yrs. Male. White. Business man.	Right thigh.	Gunshot wound.	3 yrs. 11 mos.	Arteriovenous aneurysm of femoral vessels at apex of Scarpa's triangle.	Cardiac failure; ascites; auricular fibrillation; bed ridden; treated for cirrhosis of the liver for a long time.	Reid. 7/12/28	Excision of arteriovenous fistula; quadruple ligation of femoral vessels.	Well.	Case reported in detail in the Am. Jr. of Surg., 1931; also Matas Birthday Volume. This patient was so sick he had to be operated upon in the sitting position and under local anesthesia. He is still going about and attending to his business.
7	9/6/30 C. G. H. No. P-10080	26 yrs. Male. White. Farmer.	Left lower thigh.	Gunshot wound.	1 mo.	Arteriovenous aneurysm of femoral vessels in Hunter's canal.	Swelling and bruit and thrill; impaired circulation to foot.	No operation.	None.	Unknown.	This patient was to return for operation after collateral circulation developed. It has been impossible to locate him. No noticeable change in heart during the month of observation.
8	3/10/31 Holmes Hospital. No. 310103	25 yrs. Male. White. Clerk.	Left groin.	Gunshot wound.	17 yrs.	Arteriovenous fistula of femoral artery and vein at the level of the profunda.	Shortness of breath; irregular heart action; cramps in his leg; thrill and bruit.	Reid. 3/14/31	Excision of fistula; quadruple ligation of vessels and profunda artery.	Well.	This case illustrated the extent of the cardiac damage resultant from a big fistula and the circulatory embarrassment of the extremity beyond it. His heart has returned to normal size and function. This case is reported in detail in the ANNALS OF SURGERY, 95, 578-580, April, 1932. March, 1938, patient entirely well.
9	3/20/31 Children's Hospital. No. 7375	1 yr. Female. White. Child.	Right lower extremity.	Congenital.	Since birth.	Extensive cirsoid aneurysm.	Marked enlargement and overgrowth of leg; large ulcer of calf; strong thrill and loud bruit.	Reid. 1. 3/12/31 2. 6/11/31	1. Ligation of femoral artery and vein. 2. Mid thigh amputation.	Well.	This case is reported in detail in the J.A.M.A., 191, 1391, Oct. 28, 1933. No recurrence in stump in June, 1937.
10	6/27/31 Holmes Hospital. No. 310278	23 yrs. Female. White. Physician.	Right posterior chest wall.	Thoracentesis.	5 yrs.	Arteriovenous aneurysm of the 9th intercostal vessels.	Bruit and very faint thrill.	No operation.	None.	Well.	This lesion was accidentally discovered about 3 years after the thoracentesis. By 1931, the area of audible arteriovenous bruit was 9 x 7 cm., with maximum intensity about 7 cm. from the 9th dorsal spine. After 1931, the signs of the aneurysm began to diminish and on the last examination, in the fall of 1937, only a very faint systolic bruit could be heard over a small area.
11	4/30/32 Baptist Hospital. Birmingham.	56? yrs. Female. White. Housewife.	Neck and occipital region.	Spontaneous; following tonsillitis and influenza.	1 yr.	Cirsoid aneurysm.	Noise in head; nervousness.	Reid. 5/1/32	1. Excision of occipital vessels. 2. Ligation of external carotid artery.	Slight relief for few days. Complete return of symptoms.	Patient operated upon twice on the same day. Following excision of occipital vessels and fistulae, the bruit returned in 2 hours. The same day, the wound was reopened and the external carotid artery ligated.
12	5/30/32 Booth Memorial Hospital.	50? yrs. Male. White. Cobbler.	Right popliteal space.	Gunshot wound.	4 mos.	Arteriovenous fistula of popliteal vessels.	Peroneal nerve palsy; some shortness of breath; no pulse in foot.	Reid. 5/27/32	Matas occlusive endoaneurysmorrhaphy under tourniquet.	Well.	Pulse dropped 16 beats and the blood pressure went up 14 points on occlusion of fistula. Heart not perceptibly damaged, either clinically or by roentgenologic studies. Rather alarming hemorrhage on removal of tourniquet. Patient still has peroneal palsy, due undoubtedly to the bullet. Circulation in leg is good.
13*	8/9/32 Massachusetts General Hospital. No. 29001	32 yrs. Male. White. Merchant.	Right popliteal space.	Puncture of leg with point of scythe.	17 yrs.	Arteriovenous fistula of popliteal vessels.	Varicose veins; increase in size and length of leg; ulcer on shin; partial foot-drop.	1. A. W. Allen 5/8/31 2. Reid. 8/9/32	1. Ligation of popliteal artery. 2. Quadruple ligation and occlusion of fistula in situ.	Well.	Following ligation of the popliteal artery this patient had marked circulatory embarrassment as evidenced by chronic ulcer and partial foot-drop. It was necessary for him to go on crutches for a year. Following second operation, circulation improved markedly; ulcer healing; paralysis disappeared; and in a short time there was no disability.
14	12/31/32 C. G. H. No. Q-19776	16 yrs. Female. Colored. School.	Right side of neck.	Gunshot wound.	1 mo.	Arteriovenous fistula of right carotid vessels at base of skull.	Noise in head; bruit and thrill over carotid vessels.	No operation.	Rest in bed for one month.	No improvement.	Patient sent home to return for operation later. It has been impossible to get in touch with her since then. During the month of observation no serious cardiac symptoms developed.
15	2/11/34 Holmes Hospital. No. 340079	23 yrs. Male. White. Unemployed.	Right popliteal space.	Gunshot wound.	2 yrs.	Arteriovenous aneurysm of popliteal vessels.	Pain and swelling of leg with an ulcer on shin; precordial pain; palpitation; dyspnea; thrill and bruit.	Reid. 2/15/34	Excision of arteriovenous fistula; quadruple ligation of vessels.	Well.	This case was reported by Dr. Johnson McGuire in the Am. Heart Jr., 10, 360, February, 1935. Heart now appears to be entirely normal in size and action. Ulcer of leg healed. No edema. Good arterial circulation. A few varicose veins persist. Wears an elastic stocking.
16*	6/18/34 Holmes Hospital. No. 340334	24 yrs. Female. White. School teacher.	Right forearm.	Congenital.	Noted by parents at age 3.	Cirsoid aneurysm involving volar surface of right forearm and hand.	Marked disability in use of hand; discomfort and tightness in the forearm; fear of hemorrhage.	Reid. 6/19/34	Excision of pulsating angiomatous condition from the muscles, tendons and nerves of right forearm and hand.	Well.	It was necessary to sacrifice the muscle of the flexor sublimis tendon to the index finger. This tendon was sutured into the corresponding tendon to the third finger. The angioma contained many phleboliths, which show in the roentgenogram. The patient now has perfect function and normal strength in her hand.
17	C. G. H. 4/23/34 No. 22221 7/5/34 No. 25581 10/22/34 No. 30502 5/10/35 No. 39720 5/23/36 No. 56617 10/11/37 No. 80852	34 yrs. Male. Colored. Laborer.	Entire left arm and axilla.	Congenital.	Observed by patient 6 yrs.	Cirsoid aneurysm.	Swelling, pain and disability of left arm; increase in size and length of arm (1 inch).	Herrmann. 1. 10/31/34 2. Reid. 11/23/34 3. 10/14/37	1. Multiple ligations of and excision of superficial veins of arm. 2. Same. 3. Incision and drainage of axillary, infected hematoma.	Improved.	Venous pressure, left 14.5; right 5 cc. water. Surface temperature left arm markedly elevated. Bones normal. Blood pressure left 185/110; right 150/80. Although lesion was congenital, acute onset of trouble only 6 years following extensive thromboses. Further ligations and sclerosing injections are being undertaken. Arteries of arm very large, shown by arteriograms. CO ₂ venous blood left arm 44 vols. %; right 50 vol. %. Wassermann positive. Oscillations of left arm markedly increased. No increase in size of heart.
18*	11/10/34 Holmes Hospital. No. 340642	35 yrs. Female. White. Cosmetician.	Left axilla.	Gunshot wound.	7 mos.	Arteriovenous fistula between axillary vessels.	Swelling of arm; marked paralysis of hand; noise in axilla.	Reid. 11/22/34	Excision of axillary artery and vein including the fistula; freeing of nerves from scar tissue.	Well.	Patient previously operated upon one month after the fistula by another surgeon. At that operation one fistula was closed but the symptoms promptly returned. The patient now has complete use of her hand and arm.
19	6/22/35 Children's Hospital. No. 16124	15 yrs. Male. White. Child.	Lower left thigh.	Blow over lower inner left thigh.	6 yrs.	Cirsoid aneurysm.	Swelling of inner side of lower thigh; bruit; numbness of leg.	Reid. 6/24/35	Excision of femoral artery and vein; quadruple ligation in Hunter's canal.	Improved.	Patient operated upon twice before in Wichita, Kan.; first in 1931, when the bone was exposed; again in June 1933, when the femoral artery was ligated. At the time of my operation, condition resembled cirsoid aneurysm, and there appeared to be numerous fistulae. Reexamination, in June 1936, revealed the presence of thrill and bruit over the femoral vessels in the groin, but not at site of operation. February 18, 1938, entire leg involved, especially ankle and groin where there are large superficial blood vessels, thrill and bruit. Faint bruit, no thrill, at site of operation.

17	4/23/34 No. 22221 7/5/34 No. 25581 10/22/34 No. 30502 5/10/35 No. 39720 5/23/36 No. 56617 10/11/37 No. 80852	34 yrs. Male. Colored. Laborer.	Entire left arm and axilla.	Congenital.	Observed by patient 6 yrs.	Cirroid aneurysm.	Swelling, pain and disability of left arm; increase in size and length of arm (1 inch).	Herrmann. 1. 10/31/34 2. 11/23/34 3. 10/14/37	1. Multiple ligations of and excision of superficial veins of arm. 2. Same. 3. Incision and drainage of axillary, infected hematoma.	Improved.	Venous pressure, left 14.5; right 5 cc. water. Surface temperature left arm markedly elevated. Bones normal. Blood pressure left 185/110; right 150/80. Although lesion was congenital, acute onset of trouble only 6 years following exten- sive thromboses. Further ligations and scler- osizing injections are being undertaken. Arteries of arm very large, shown by arteriograms. CO ₂ venous blood left arm 44 vols. %; right 50 vol. %. Wassermann positive. Oscillations of left arm markedly increased. No increase in size of heart.
18*	11/19/34 Holmes Hospital. No. 340642	35 yrs. Female. White. Cosmetician.	Left axilla.	Gunshot wound.	7 mos.	Arteriovenous fistula between axil- lary vessels.	Swelling of arm; marked paralysis of hand; noise in axilla.	Reid. 11/22/34	Excision of axillary artery and vein including the fistula; freeing of nerves from scar tissue.	Well.	Patient previously operated upon one month after the fistula by another surgeon. At that operation one fistula was closed but the symptoms promptly returned. The patient now has complete use of her hand and arm.
19	6/22/35 Children's Hospital. No. 16124	15 yrs. Male. White. Child.	Lower left thigh.	Blow over lower inner left thigh.	6 yrs.	Cirroid aneurysm.	Swelling of inner side of lower thigh; bruit; numbness of leg.	Reid. 6/24/35	Excision of femoral artery and vein; quadruple ligation in Hunter's canal.	Improved.	Patient operated upon twice before in Wichita, Kan.; first in 1931, when the bone was exposed; again in June 1933, when the femoral artery was ligated. At the time of my operation, condition resembled cirroid aneurysm, and there appeared to be numerous fistulae. Reexamination, in June 1936, revealed the presence of thrill and bruit over the femoral vessels in the groin, but not at site of operation. February 18, 1938, entire leg involved, especially ankle and groin where there are large superficial blood vessels, thrill and bruit. Faint bruit, no thrill, at site of operation.
20	8/27/35 C. G. H. No. 44516	28 yrs. Female. White. Laundress.	Intracranial.	Blow on angle of right lower jaw.	2 wks.	Arteriovenous fistula between in- ternal carotid artery and caver- nous sinus.	Pulsating exophthalmos; diplopia; thrill and bruit; pulsation of retinal veins.	Zininger. 9/13/35	Ligation of external carotid artery with braided silk; common carotid with metallic band.	Improved.	December 1, 1935: Patient working steadily but still some exophthalmos, and a faint bruit and slight diplopia. Unable to trace patient since.
21*	5/13/36 C. G. H. No. 56203	47 yrs. Male. White. Laborer.	Left subclavian region.	Gunshot wound.	14 mos.	Arteriovenous aneurysm left sub- clavian artery and vein.	Partial paralysis of left hand.	No operation.	Bed rest.	Spontaneous healing at end of 14 mos.	This patient was not operated upon because of extensive pulmonary tuberculosis and syphilis. The thrill and bruit became gradually less and finally all signs of the fistula disappeared. Months after the accident, the paralysis was mainly of the ulnar nerve and this, too, improved rapidly after the closure of the fistula.
22	9/16/36 C. G. H. No. 61788	14 yrs. Female. White. School.	Left forearm and hand.	Congenital.	Since birth.	Cirroid aneurysm.	Marked swelling of forearm and hand with very large superficial veins; bruit; marked disability on use of hand; overgrowth of arm.	Reid. 9/29/36	Excision of aneurysm from muscles, tendons and nerves of forearm and hand.	Improved.	It was impossible to perform a complete excision because of the infiltration in the bellies of the muscles and between the bones. Following the operation some sclerosing injections were intro- duced into the large vessels of the dorsum of the hand and fingers. There is marked improvement in the function of the hand. Operation was per- formed with arm suspended straight in the air to lessen hemorrhage.
23*	10/20/36 C. G. H. No. 63741	53 yrs. Male. Colored. Laborer.	Left side of neck.	Gunshot wound.	3 yrs.	Arteriovenous aneurysm of internal carotid artery and jugular vein at base of skull.	Ring in the ear; dizziness; pulsation in neck.	Reid. 11/6/36	Closure of fistula by dividing and twisting jugular vein.	Well.	Patient died February 11, 1937, from extensive bilateral tuberculosis of the lungs. Specimen of vessels obtained. Fistula closed.
24*	4/16/37 C. G. H. No. 73329	30 yrs. Male. Colored. Laborer.	Left femoral region.	Gunshot wound.	15 yrs.	Arteriovenous aneurysm of femoral vessels.	Cardiac decompensation; ascites; hy- drothorax; edema of extremities.	Reid. 5/8/37	Division of femoral artery and vein; transfixion of communication.	Well.	Patient was orthopedic; numerous taps for hydro- thorax; operation under local anesthesia, with the patient in Fowler's position; marked improve- ment on table when fistula was closed. Has re- sumed work.
25*	4/16/37 C. G. H. No. 73338	19 yrs. Male. White. Laborer.	Right side of neck and face.	Congenital.	Since birth.	Pulsating mass below left ear as long as can remember.	Slight roaring in ear; swelling.	Reid. 5/4/37	Ligation of external carotid artery; double ligation of occipital; divi- sion and twisting of external jugu- lar vein.	Well.	This patient had multiple communications be- tween external jugular vein and branches of ex- ternal carotid artery, mainly a large occipital artery.
26	6/13/37 C. G. H. No. 25847	24 yrs. Male. Colored. Laborer.	Right femoral region.	Gunshot wound.	Immediate.	Arteriovenous aneurysm between the femoral vessels at Poupart's ligament.	Hematoma; typical thrill and bruit.	No operation.	None.		This patient was discharged to wait for collateral circulation before operating. Unable to get in touch with him since then.
27*	9/10/37 Christ Hos- pital. No. 112543	33 yrs. Male. White. Laborer.	Left arm.	Compound fracture of humerus.	16 yrs.	Arteriovenous aneurysm of brachial vessels, just above elbow.	Shortness of breath; palpitation; nerv- ousness; slight cardiac enlargement.	Zininger. 9/11/37	Excision of fistula; quadruple liga- tion of the vessels.	Well.	Heart decreased in size. Blood and plasma vol- umes decreased approximately 25% after opera- tion; circulation time became 4 to 5 seconds longer. All complaints disappeared.
28*	10/22/37 C. G. H. No. 81335	16 yrs. Male. White. School.	Right arm just above elbow.	The cap of a 25- caliber bullet.	19 das.	Arteriovenous aneurysm of brachial artery and vein.	Partial paralysis of radial nerve; very thin skin over localized swelling.	Reid. 10/26/37	Excision of fistula; quadruple liga- tion of vessels.	Well.	Patient was operated upon early because of the rapid, progressive paralysis of the radial nerve, and the fear of rupture of the aneurysm. Two veins, as well as the artery were involved. No circulatory disturbance after operation. Paraly- sis of the nerve has disappeared (February 15, 1938).
29*	10/10/37 2/10/38 C. G. H. No. 80815	18 yrs. Male. White. Laborer.	Right popliteal space.	Gunshot wound.	4 mos.	Arteriovenous fistula between pop- liteal artery and vein.	Marked thrill, bruit and enlargement of femoral artery; embarrassment of circulation to foot.	Reid. 2/16/38	Excision of fistula; quadruple liga- tion.	Well.	Slight cardiac damage. Pulse in foot reappeared after excision of the aneurysm. Blood volume not increased during the four months. Branham's bradycardic phenomenon present; also, eleva- tion of blood pressure on closure of the fistula.
30*	2/9/38 C. G. H. No. 88134	19 yrs. Male. White. Farmer.	Left foot, fore- arm and neck.	Spontaneous.	10-12 yrs. in arm; 3 or 4 yrs. in neck and left foot.	Multiple (4) cirroid aneurysms.	Slight huskiness of voice; congestion of face.	Reid. 1. 2/12/38 2. 2/26/38 3. 4/26/38	1. Excision of cirroid aneurysm of arm and foot. 2. Partial excision of cirroid aneu- rysm of neck. 3. Ligation right external carotid artery.	Arm and foot well; neck markedly improved.	Definite cardiac enlargement with murmur. Com- munications were definitely demonstrated at time of operation. Immediately after ligating the right external carotid artery (4/26/38) bruit markedly decreased, but still faintly audible.

* Reported in detail in this paper.

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and the heart. This instrument, based upon well known laws of physics, measures the velocity of flow regardless of the pressure of the fluid. From this data the volume flow may be determined.³ In five experiments direct anastomoses were made between the abdominal aorta and vena cava. In our chronic experiments we have in the laboratory two dogs with large fistulae between the iliac artery and vein.

A summary of our findings in the acute or sacrifice experiments is presented in Chart 1. In three dogs the cardiac output, as determined by the direct Fick method, increased an average of 1.75 liters when the aortic-vena caval fistula was open. This was an increase of over 100 per cent. Altogether, six determinations were made upon these three dogs.

The circulation time was determined by the sodium cyanide method.⁴ When this drug was injected into the vena cava about one inch above the fistula, the circulation was *reduced* an average of 4.5 seconds, whereas, the circulation time in the femoral vein, below the fistula, *increased* by an average of 3.9 seconds. Altogether 13 determinations were made upon the two dogs.

The venous pressures measured in the femoral vein increased on an average of 16.8 cm. of water, whereas, two inches above the fistula (*i.e.*, between fistula and heart), there was an average increase of only 3.8 cm. of water. These figures were based upon eight determinations made upon the four dogs. Inasmuch as the rise of venous pressures above the fistula was so slight and quite contradictory to the findings of Holman⁵ and Ney,⁶ we wondered if the venous pressure changes might be seriously altered by the rubber tube which formed the fistula between the aorta and vena cava. Consequently, some direct anastomoses were made just above the bifurcation of the aorta, and venous pressures, determined through the renal vein, were essentially the same as when the rubber tube was used for the connection of the aorta and vena cava. In no instance were we able to obtain the high venous pressures, well above the fistula, as were reported by Holman and Ney. The

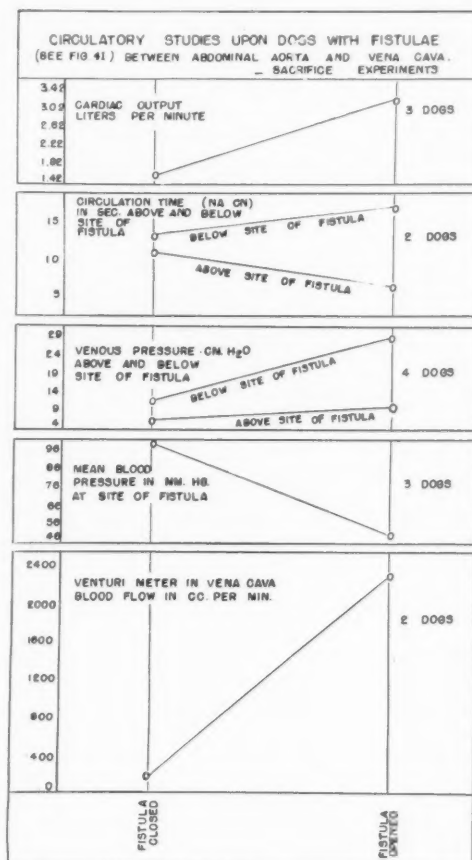


CHART 1

tremendous increase in cardiac output may, we think, explain the low venous pressures we obtained. Below, or distal to the fistulae, our observations were similar to those of other observers.

The mean blood pressure in the artery at the site of the fistula dropped an average of 49.6 Mm. Hg. in 12 determinations upon three dogs.

In two dogs, 13 measurements were made with the Venturi meter (Chart 1) of the amount of blood flow in the vena cava, both when the fistula was closed and open. There was an average increase of 2,090 cc. per minute through the vena cava above the fistula, when it was open. So far as we know, this is the first time the actual amount of blood flowing through this segment of vein has been measured in both the presence and absence of a fistula between the aorta and vena cava lower down. The amount of the increase of blood flow, when the fistula was open, was astounding to us, and when the aorta just distal to the fistula was temporarily occluded, the increase of the blood flow in the vena cava was far greater still.

There were a few other observations, perhaps worthy of note, in addition to those which are charted. For instance, whenever the fistula was opened, there was uniformly an increase of about 20 per cent in the heart rate. In one animal when all the aortic blood was diverted back through the vena cava into the heart, a systolic cardiac murmur developed in about five to seven minutes, and this disappeared in a similar period when the fistula was again closed. If, under this circumstance, the fistula was not closed shortly after the development of the murmur, cardiac failure seemed imminent.

By means of teleoroentgenograms we could not demonstrate any decrease in the size of the heart when the fistula was open. On the contrary, when the hearts of these dogs were exposed, all observers agreed that the heart, especially the right ventricle, became larger within less than a minute after opening the fistula. This increase was, also, roughly confirmed by measurement with calipers. Nor in our survival experiments have we been able to demonstrate any temporary decrease in the size of the heart after making the fistulae. These observations do not coincide with those of Holman, who noted decreases in the size of the heart for several hours or days after producing large arteriovenous fistulae in dogs, but do coincide with those of Lewis⁷ who, after the lapse of a few beats, noted a steady increase in the size of the heart.

In the two chronic or survival experiments with large iliac arteriovenous fistulae, the usual changes have been noted—cardiac enlargement, Branham's bradycardic phenomenon, rise of blood pressure on closure of the fistula, *etc.* The venous pressures in the limb below or distal to the fistula have remained constantly elevated, whereas these pressures in the neck have not increased since the fistulae were made. Also, in this connection, it should be noted that not as yet (Exper. 1: 18 weeks' duration; Exper. 2: nine weeks' duration) have there developed any evidences of cardiac decompensation.

Up to this point, the blood volumes, as determined by brilliant vital red dye, have not increased. The blood volume made before operation in Dog

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No. 1 was 1,955 cc.; since operation, 1,781 cc.; before operation in Dog No. 2, 1,815 cc.; since operation, 1,745 cc.

Discussion of Clinical and Experimental Observations.—The 30 cases may be classified as follows: (1) *Arteriovenous aneurysms, 21 cases.* Of these there were six femorals, four popliteals, three brachials, two internal carotids, two intracranials (pulsating exophthalmos), one axillary, one subclavian, one ophthalmic, and one intercostal. (2) *There were nine cirroid aneurysms* which may be classified as to location as follows: Neck, three; leg and foot, three; arm and hand, three. One of these cases (No. 30) had four spontaneous cirroid aneurysms but is classified as one neck case because of the situation of the major lesion. In this case there were two other cirroids in the left forearm and one on the dorsum of the left foot.

Six of these cases were never operated upon. In the 24 cases which were operated upon there was a total of 39 operations. In the entire group there was not a death which could be attributed to the aneurysm or the operative procedures. It is known that Case 2 died from pulmonary tuberculosis two and one-half years after the operation, and Case 22, from the same cause, three and one-half years later. In both instances the aneurysms were cured. All of the arteriovenous aneurysms operated upon, except one case (No. 20) of pulsating exophthalmos, were cured, and two cases (Nos. 10 and 20) healed spontaneously without operation. Of the nine cases of cirroid aneurysm there appear to be only three complete cures (Nos. 9, 16 and 25), and one of these cures was brought about by amputation. All of the others have been benefited to varying degrees and none of them have lost an extremity.

A study of all of these cases and the results of our experimental work lead us to make some comments upon the effects, and the treatment, of arteriovenous and cirroid aneurysms. In most instances this work confirms previous observations reported by Halsted,⁹ Matas,¹⁰ Holman,⁵ Lewis,⁷ Reid¹ and many others. In view of the recent complete bibliographies given by Holman and others, reference to the extensive literature on this subject will be made only when our conclusions and observations are at variance with those of other investigators.

Damage to the Heart.—The causal relationship between arteriovenous and cirroid aneurysms and cardiac damage has been definitely established since Reid first observed it experimentally, in 1918. Holman's splendid work in this connection no longer leaves room for doubt. The main effects of the fistulae are cardiac enlargement, principally of the right ventricle, and eventual decompensation. The degree of cardiac damage is directly dependent upon the vessels involved and the size of the fistula; the larger the vessels and the bigger the fistula, the greater is the damage to the heart. In other words, the size of the spillway and the amount of arterial blood spilled directly over into the vein seem to be the major determining factors. The effects upon the heart of the altered blood pressures and the proximal dilatation of the artery have not been definitely determined. The heart, in the chronic cases, is rarely affected seriously unless the artery between the fistula and heart is definitely

dilated, and one cannot escape the feeling that whatever is responsible for this change in the artery may also be effective upon the heart.

In this series of cases there were eight arteriovenous aneurysms which had caused definite cardiac damage (Nos. 3, 6, 8, 15, 21, 24, 27 and 29). In two cases (Nos. 6 and 24) there was severe cardiac decompensation. Of the cases of cirroid aneurysms two (Nos. 17 and 30) showed evidences of some cardiac damage. In every instance where the heart was demonstrably affected, closure or excision of the fistula was followed by improvement in the condition of the heart. In Case 24 (Fig. 7) the reduction in the size and the improvement in the function of the heart were quite astounding.

The Effect of Fistulae Upon Involved Vessels.—The "venafication" of the artery between the fistula and heart is exemplified by the thinning of its wall, tortuosity and marked dilatation. Indeed, in some of these proximal vessels there have been reported true arterial aneurysms. In these vessels there was a great fall in systolic blood pressure. This reduction of strain or work upon the vessel wall probably accounts for its atrophy, which is evident in microscopic sections. Perhaps the blood pressure alterations reduce the nourishment to the vessel wall through the vasa vasorum. In any event, the atrophy of the proximal artery appears to confirm Thoma's old theory that a normal pulse pressure is essential to the integrity of an artery.

The "arterialization" or hypertrophy of the involved vein is logically explained by its increased work and adaptation to heightened pressures. That the vein between the fistula and heart carries a tremendous increase in volume of blood is shown by our studies with the Venturi meter upon dogs, where the average increase in volume of blood flowing through the vena cava between the aortic-vena caval fistula and heart was 2,090 cc. of blood (Chart 1). From inspection it appears that very little true venous blood can find its way back to the heart through this segment of vein when the fistula is open; the force of the arterial spillway appears to let very little of the venous blood pass by the fistula.

In the artery and vein opposite the fistula extensive calcification was noted in the walls of the vessels. The explanation for this is not obvious to us.

In this series of cases there were 11 (Nos. 3, 6, 8, 13, 15, 17, 23, 24, 25, 27 and 29) which showed definite enlargement, and thinning of the wall of the proximal artery. In Case 24, where the proximal artery was so very large and thin-walled, there appeared to be imminent danger of rupture of the artery proximal to its ligation. In many cases the wall of the involved vein was definitely hypertrophied.

The Circulation Time.—Studies of the circulation time, employing the sodium cyanide method, were made upon six patients (Nos. 15, 21, 25, 27, 28 and 29) and on all of our experimental dogs (Chart 1). A very striking finding, which was not unexpected, was an acceleration of the rate when the drug was injected into the vein between fistula and heart, and a very definite retardation when it was injected on the opposite or distal side of the fistula. When the drug was injected into veins in parts of the body far removed from

or not directly affected by the fistula, there was no positive evidence that the circulation time was definitely affected by the fistula; in a few cases (Nos. 27, 28 and 29) it was about three seconds faster when the fistulae were present than it was after their extirpation, while in Case 25 it was four seconds slower than it was after operation. However, no circulation times were made upon patients with cardiac decompensation resulting from arteriovenous aneurysms. In the one case (No. 24) the patient was so sick we were loath to use the test. Similar studies have been reported by Porter.¹⁵

Blood Volume.—Blood volume determinations were made upon Cases 25, 27 and 29, and in the case of our two survival experiments upon dogs. In Case 25 there was a drop of 1,000 cc. 11 days after the operation, but nine months after the operation it was practically the same as before operation. In Case 27 there was a drop of 600 cc. in blood volume 13 days after the operation and there have been no determinations since then. In Case 29 there was an increase of 400 cc. three weeks after the operation. In all of the observations upon dogs, even though the heart has definitely enlarged, there has been no increase in blood volume; in fact, there has been a decrease of 174 cc. in Dog No. 1 and of 70 cc. in Dog No. 2. In none of the patients upon whom these studies were made was there congestive heart failure.

Our studies, thus far, fail to confirm the observations of Holman⁵ and others, who have reported a large increase in blood volume in cases of arteriovenous aneurysms. That an increase would occur in cases of heart failure due to arteriovenous fistulae should be expected, inasmuch as Gibson and Evans¹¹ have shown it to be the case in all instances of congestive heart failure regardless of the cause. But the necessity for its occurrence in cases of arteriovenous fistulae, without heart failure, seems to us questionable. The increase of pulse rate and the enormous increase of cardiac output are quite sufficient to explain the cardiac damage, without the necessity of postulating an increased blood volume. Besides, we doubt the accuracy of the dye method of determining blood volume in these cases, inasmuch as the normal dissemination or dilution of the dye must be markedly altered by the presence of a large arteriovenous fistula.

Branham's Bradycardic Phenomenon.—The slowing of the pulse rate, when the fistula was closed, was noted in ten cases (Nos. 6, 8, 12, 13, 15, 18, 24, 27, 29 and 30) and in all of our experimental animals. The extent of this slowing of pulse rate varied greatly and seemed to be directly related to the seriousness of the cardiac damage and the size of the fistulae. For instance, in Case 24, where there was very serious cardiac failure (Fig. 7), the pulse rate dropped from 80 before operation down to 40 three hours after it. The cause of this phenomenon, as so well discussed by Holman, is undoubtedly related to the sudden rise in blood pressure which either reflexly or directly stimulates the cardio-inhibitory center of the brain, or possibly has a direct effect upon the myocardium.

Venous Blood Pressures.—Observations upon the venous pressures were made in nine of our cases (Nos. 8, 15, 17, 21, 24, 25, 27, 28 and 29). When

these pressures were taken in parts of the body far away from the direct or local effects of the fistula, there was no noticeable effect upon the general venous pressures as long as there was no cardiac decompensation. For instance, when observations were made in the arm, in the case of a femoral arteriovenous aneurysm, there was no appreciable change in the venous pressures on closing the fistula or following its operative cure. However, in Case 24 with a femoral arteriovenous aneurysm, when there was severe cardiac decompensation, the venous pressure in the right arm was 25 cm. of water and was unaffected by temporary closure of the fistula; after operation and when the heart was completely compensated, it dropped to 5 cc. of water, or normal. Thus it would seem a logical conclusion from our studies that the general venous pressures are unaffected by arteriovenous aneurysms unless there occur some evidences of cardiac decompensation, when the changes in venous pressures are similar to those which occur in cases of cardiac decompensation from any other cause. We would feel that a marked rise in the general venous pressures might be considered evidence of impending myocardial failure even though this was not obvious clinically.

Our studies of venous pressures in an extremity above and below the site of a fistula do, perhaps, deserve some special comment in view of the reports by Holman and Ney that such pressures are markedly increased in the vein proximal to the fistula. In Case 29, a right popliteal arteriovenous aneurysm, the venous pressure in the femoral vein, as measured by the direct method, was $5\frac{3}{4}$ cm. of water with the fistula open, and $4\frac{1}{2}$ cm. when it was closed. At the same time the venous pressure in both antecubital veins was 10 cm. of water. Three weeks after the operation the venous pressures in these same three vessels were 7.75 cm. of water. In several cases the venous pressures distal to the site of fistula were markedly elevated as long as the fistula was open and promptly dropped to the general normal level when the fistula was closed or cured. These clinical observations coincide exactly with our experimental studies (Chart 1). This very slight rise of venous pressure between the fistula and heart was an unexpected finding. Perhaps the enormous increase in cardiac output (Chart 1) relieves the pressure within the vein as long as the heart is compensating for the increase in amount of blood it has to handle.

Blood Pressure Changes.—A rise in both systolic and diastolic pressures following closure of arteriovenous fistulae was noted in 13 cases (Nos. 6, 8, 12, 15, 17, 18, 23, 24, 25, 27, 28, 29 and 30). In some cases this rise was quite striking, as in Case 24, where it rose from 140/40 to 190/90 almost immediately on temporarily closing the fistula. Following operation this increase of the arterial pressures gradually falls until at the end of ten to 14 days it becomes stabilized or normal. The permanent rise of the diastolic pressure is more striking than the alteration of the systolic, which may ultimately return to the preoperative level.

In three cases with fistulae in the neck (Nos. 23, 25 and 30) there were very pronounced elevations of the arterial pressures during dissections of the

carotid sinus, even before anything had been done to the arteriovenous fistula. In Case 23, during manipulation of the carotid sinus the pressure rose from 120/100 to 170/110. In no case was there a permanent rise in blood pressure; in two cases the pressures returned, after two weeks, to levels slightly below the preoperative values.

Collateral Circulation.—There were six cases (Nos. 6, 8, 13, 15, 24 and 29) in which our observations confirmed other clinical and experimental evidences that around an arteriovenous fistula there occurs a very extensive collateral circulation. In several cases peripheral pulses which were not palpable as long as the fistula was open could be made to appear by simple pressure closure of the fistula, and after operation they appeared and remained good. Curiously enough, the pulse may appear, for the first two or three postoperative days, to be stronger than in the opposite extremity. After this there is a gradual and definite decrease in the volume of these pulses for a period of about ten days.

It is this overabundant collateral circulation which makes the occurrence of gangrene after excision of chronic arteriovenous fistulae practically unknown.

Impairment of Peripheral Circulation.—The circulation, considerably distal to the fistula, may be markedly impaired as evidenced by absent pulses, coldness of the part, cyanosis and occasional chronic ulcers which will not heal. This impairment, in varying degrees, was noted in five cases (Nos. 6, 8, 12, 13 and 15). In Case 15, a popliteal arteriovenous aneurysm, there had been a chronic ulcer of the shin for a long time and occasionally there had occurred serious bleeding from it.

Effect on the Growth of Extremities.—In the region of arteriovenous fistula, especially in those cases of cirroid aneurysms where there are multiple fistulae, there appears to be increased circulation. In addition to an elevation of surface temperature and the known elaborate collateral circulation, another evidence is the occasional increase in the length of an extremity when the fistulae are present before the time of the ossification of the epiphyses. This was noted in five of our cases (Nos. 5, 9, 13, 17 and 22). Naturally the most striking increases in the length and size of extremities are to be noted in the cases of cirroid aneurysms which are usually congenital, contain numerous arteriovenous fistulae and extend over large areas. This was especially observed in Case 9, which was reported in detail in the J.A.M.A., 191, 1391-1393, October 28, 1933.

Associated Nerve Paralysis.—In four cases (Nos. 12, 13, 18 and 28) there were nerve paralyses associated with the arteriovenous condition. In Case 12, this was produced by the gunshot wound and was not repaired at the time of curing the fistula. In the other three cases the paralysis seemed to be secondary to the arteriovenous aneurysms. In Case 13, we could find no injury to the peroneal nerve at the time of the operation except that it was infiltrated and distended by pulsating varicose veins (Fig. 1). That this was the probable cause of its paralysis seems to be justified by the fact that, after curing

the aneurysm, the paralysis disappeared. In Case 18, the pulsating axillary veins, together with a large amount of scar tissue, appeared to be the cause of the paralysis. After excising the aneurysm and removing most of the scar tissue, the paralysis of the arm disappeared. In Case 28, the paralysis of the median nerve was clearly due to pressure of the pulsating, false aneurysmal sac in which the arteries and veins communicated. The return of function in this nerve was very prompt after excision of the aneurysm.

Double Arteriovenous Fistula.—Occasionally the object which produces an arteriovenous aneurysm will penetrate two veins and the artery and establish a double fistula. This occurred in Cases 18 and 28. In Case 18, the surgeon who first operated cured only one of the fistulae, and it was necessary for us to operate, six months later, in order to cure the other (Figs. 8, 9, 10 and 11). In the second case the condition was recognized at the first operation and both veins and the artery were excised (Figs. 24, 25 and 26).

Arteriovenous Aneurysm Caused by Thoracentesis.—This occurred in Case 10. After about five years the characteristic bruit and thrill reached their maximum intensity, subsequent to which the signs of the aneurysm began to subside; and at the end of 13 years from the time of the injury, there were no signs of a fistula, only a faint systolic bruit indicating a slight coarctation of the intercostal artery. There never developed a pulsating, angiomatous condition of the chest wall. Roentgenologic examinations of the thorax were always normal.

Intracranial Arteriovenous Aneurysms.—There were two cases (Nos. 2 and 20) of pulsating exophthalmos, and one instance (No. 4) of a fistula between the ophthalmic vessels behind the eyeball. In Case 2 it was necessary, because of hemiplegia, to remove the aluminum band from the carotid artery eight hours after the operation. The hemiplegia promptly disappeared. Fourteen days later the band was reapplied, followed by a partial hemiplegia which entirely cleared up, except for a slight spasticity of the right arm. The aneurysm was cured. In Case 20, the external carotid artery was ligated, and the common carotid occluded by a metallic band. There were no cerebral symptoms, but the aneurysm was not completely cured at the last follow-up examination. In one case, the record of which cannot be found, the operative procedure was to divide the supra-orbital vein and to scarify the intima of the proximal part in order to enhance the chances of a propagating thrombosis which might occlude the fistula. Nothing was done to the vessels of the neck. Following this procedure, and with bed rest in a high Fowler's position and limitation of the fluid intake, there was a marked improvement, but not a complete cure.

In Case 4, arteriovenous fistula between the ophthalmic vessels, the following procedure was undertaken: (1) Ligation of the external carotid artery with braided silk. (2) Occlusion of the common carotid by an aluminum band. (3) Excision of veins from the upper eyelid and inner side of nose. Six months after this operation, a harsh bruit at the site of the band indicated that a lumen was being established in the common carotid artery. At the last

examination, April 13, 1938, the patient was entirely well. The eye was normal. At the site of the band, a faint systolic bruit indicated a good lumen beneath it. There was a strong temporal pulse which disappeared on occluding the common carotid artery. No dilatation of this artery was noted, either proximal or distal to the band; it appeared to be normal in size.

Spontaneous Healing.—In this series of cases there were two arteriovenous aneurysms which healed spontaneously. The fistula in Case 10 was between the intercostal vessels; in Case 21, between the subclavian vessels. A similar case was reported in Reid's previous series and recently Bird¹² has reported other instances. It is not an unusual occurrence in experimental fistulae between the smaller vessels.

In view of these experiences it is probably wise to use every effort to promote spontaneous healing before resorting to surgery. A long period of rest in bed, with elevation of the affected part and the limitation of fluid intake, possibly bleeding, soon after the accident, might result in more spontaneous cures. Certainly the long period of bed rest for tuberculosis was a big factor in the spontaneous healing in Case 21.

Time to Operate.—Unless immediate or early operations are required because of hemorrhage, dangerous hematoma or infection, or rapid cardiac damage, we believe it wise to postpone operating for three to six months after the occurrence of the fistula. During this period hemorrhage becomes absorbed, tissues restored to normal, danger of infection lessened, and collateral circulation becomes so extensive that there need be no hesitancy in sacrificing the involved vessels at the time of operation. Mason¹³ and Stone¹⁴ have reported two cases in which the cardiac damage developed so rapidly that they could not wait for the development of a collateral circulation. This did not occur in any of the cases reported in this paper. However, it was necessary to operate early in Case 1, because of infection and secondary hemorrhage, and in Case 28, because of impending hemorrhage and rapidly progressive median nerve paralysis. A rapidly rising general venous pressure should probably be regarded as an indication for early operation, even though the heart may not appear, clinically, to be badly affected.

During the period of time that operation is being delayed, it is our feeling that more effort should be made to improve the chances of spontaneous healing than has been made in the past. Keeping the patient in the hospital during this time not only allows the adoption of measures to promote spontaneous healing, but also is the surest way of remaining in touch with charity patients. In our series of cases we were never able to trace three patients who promised to come back for later operations (Nos. 3, 7 and 26).

Standard Curative, Operative Procedures.—The essential thing in the operative cure of arteriovenous and cirroid aneurysms is to eliminate all possibilities of any blood ever again passing through the fistulae. The procedures which have been used and which accomplish this end are:

(1) *Closure of the Fistula, with Restoration of the Vein and Artery.*—This procedure would appear, at first thought, to be ideal and physiologic.

However, in many cases this has been followed by serious pulmonary complications, due to embolisms of air, and blood clots from thrombosis at the site of the operation. Besides, the enormous venous dilatations in old cases make it unnecessary. The ligation of the involved veins probably results in a better balance between the arterial and venous beds, even though the artery is restored. This procedure was not carried out in any of our cases.

(2) *Suture of Fistula with Restoration of Artery and Ligation of the Vein.*—When the arterial wall is not atrophied and no danger is anticipated from a sudden restoration of normal blood pressure, there is no objection to this procedure. It is particularly desirable where operations are performed early, as is frequently done in Europe, and before there has been time for the development of an adequate collateral circulation. In the late cases where severe changes have occurred in the proximal artery and there is abundant collateral circulation, we do not believe in attempting to restore the artery. It is not necessary, and occasionally there develops a true arterial aneurysm after closure of the fistula. This happened in a case reported previously by Reid.¹ This procedure was not done in any of the cases reported in this series. When one elects this operative procedure, a part of the vein can often be used to advantage in closing the defect in the artery.

(3) *Quadruple Ligation of the Artery and Vein.*—This procedure is certain of succeeding only where *all* the intervening branches are ligated; otherwise, the chances of the return of the aneurysm to its previous state are excellent. In addition, this procedure carries the additional dangers of ligation in continuity as compared with those of division of the artery.

(4) *Ligation of the Canal of Communication.*—This procedure is probably dangerous and, besides, is rarely possible technically. It was done in one of Reid's early cases where the fistula was only six months old, and the canal of communication small and easily identifiable.

(5) *Extirpation of Both Vein and Artery at the Site of Fistula, with Quadruple Ligation of the Vessels.*—This was done, whenever possible, in all the cases of this series. There must necessarily be exceptions to this procedure such as in the cases of intracranial arteriovenous aneurysms, certain extensive cirroid aneurysms and, occasionally, under other circumstances which will be discussed later. Complete extirpation certainly obliterates the fistula and, in Reid's cases, has given most satisfactory results. Procedures which require the use of a tourniquet have, when possible, been avoided. For this reason we have had little experience with the Matas intrasacular restorative and obliterative procedures. In a few cases where we have elected this method of operating, the control of hemorrhage, after removal of the tourniquet, has been more troublesome than when a slow dissection was made without a tourniquet. This was especially true in Case 12 of our series.

(6) *Ligation and Division of the Involved Vessels and Transfixion Occlusion of the Fistula.*—This is a procedure which has been employed in two cases (Nos. 13 and 24; Figs. 1, 2, 3, 4 and 5), and seems to us to be an effective method of assuring the closure of the fistula when the hazards of

total ablation appear to be too great. There is no danger of hemorrhage from puncturing the vein with double braided silk after the ligations of the artery and distal vein.

(7) *Closure of the Fistula by Means of Dividing and Twisting the Vein.*—This is probably a new procedure and was effectively employed in Cases 23 and 25 (Figs. 12, 13, 14, 19, 20, 21 and 22). In these two cases it was impossible to expose the vessels distal to the point of the fistulae. In one case (Case 23) it was obvious that the patient would not tolerate a proximal ligation of the artery in addition to the twisting of the vein. Yet the autopsy, a year or more later, showed that the fistula was completely healed without any disturbance in the lumen of the artery. This technic of operating appears to us to have a definite application in some of those cases where it is not possible to use any of the other standard, curative procedures which have been discussed. Since the idea of this operative procedure occurred to us, we have not had a case of pulsating exophthalmos in which we would like to divide and free and twist the proximal portion of the supra-orbital vein in addition to whatever else might be undertaken in the neck.

Palliative Operative Procedures.—In old cases, when the heart is badly damaged, there may be some question as to whether the heart can stand the physiologic change incident to a sudden closure of the fistula. Both Matas and Holman have spoken of this and have advocated, and cite, the employment of such procedures as repeated temporary digital compressions of the fistula in order gradually to accustom the heart to a permanent closure of the fistula. In none of the cases in this series did it appear necessary to adopt these preoperative procedures, although in Case 24, there occurred a rather alarming bradycardia after the operation and, at operation, the proximal artery came near rupturing when it was ligated.

In one of Reid's cases, reported in 1925, Halsted ligated the proximal vein with great relief to the heart. When the measures advocated by Matas leave a real doubt as to whether the heart can stand a sudden extirpation of the fistula, it might be well to consider preliminary ligations of the proximal, or even the proximal and distal vein, before performing the operation which will permanently close the fistula.

Mistakes in Operative Procedures.—In general, any procedures which do not actually close the fistula are undesirable and run serious risks of not only failing to cure the aneurysms, but of causing serious circulatory disturbances peripheral to the fistulae. An untold number of limbs have become gangrenous and have had to be amputated because of the *simple proximal ligation of the artery*. It is far more dangerous than the ligation of an artery for an arterial aneurysm, for the shunt or spillway remains and there is no longer enough arterial force to push the blood beyond it. Although all authors discussing this subject in recent years have severely criticized the proximal ligation of the artery for arteriovenous aneurysms, it is still unnecessarily done. In Case 13 (Fig. 1), the patient was made an invalid for over a year and almost lost his leg following a proximal ligation of the artery.

In the preceding paragraph we have used the word "unnecessarily," for there are some abnormal arteriovenous communications for which it still seems necessary to take the risk of performing a proximal ligation of the artery. In the case of the extensive cirroid aneurysm, where a direct attack upon the numerous fistulae is impossible, there is apparently no risk in ligating almost as many proximal arteries and veins as one can find (Cases 5, 9 and 10). Again in cases of pulsating exophthalmos (Nos. 2 and 20), and in such a case as Case 4, it would appear better to take the chances of a proximal ligation than to carry out the direct attacks upon the fistulae. In addition to ligating the external carotid and occluding the common carotid with a removable metallic band, we shall in the future try dividing and twisting the supra-orbital vein as has already been suggested. The ligation of the jugular vein, as advocated by Holman, should probably also be done.

SUMMARY

(1) An analysis of 21 cases of arteriovenous and nine cases of cirroid aneurysms is presented, which is supplemented by observations upon experimentally produced arteriovenous aneurysms in dogs.

(2) Sixteen of the arteriovenous aneurysms were operated upon and all of them, except one case of pulsating exophthalmos, were cured. In two instances the aneurysms healed spontaneously without operation. Four patients failed to return for later operations and could never be traced. All of the nine cirroid aneurysms were operated upon; three were cured and the other six were more or less improved. There were no deaths in the entire series of 30 cases. There was a total of 39 operations upon the 24 patients who were subjected to surgical treatment.

(3) Clinical and experimental observations which may throw some light upon the physiologic and pathologic effects of arteriovenous fistulae are discussed in some detail. The principal effects noted and studied were: Ten instances of cardiac damage; 11 instances of thinning and dilatation of the proximal artery; circulation time upon six patients; blood volume upon three patients; ten instances of Branham's bradycardic phenomenon; 13 instances of blood pressure alterations; studies upon the venous blood pressures of nine patients; nine instances of markedly increased collateral circulation; five instances of impairment of the circulation peripheral to the fistula; five instances of an increase in the size and length of an extremity; four instances of associated nerve paralyses; two instances of double arteriovenous fistulae; and two instances of spontaneous healing of the aneurysm.

(4) In our limited clinical and experimental observations, we could not confirm Holman's findings of a marked increase of the total circulating blood.

(5) A Venturi meter was used in some of the experiments to measure the flow of blood in a segment of the vena cava. An easy method of making an arteriovenous fistula which can be alternately closed and opened is illustrated.

(6) The time to operate, and the standard curative operative procedures, are discussed. Two new operative procedures are illustrated and described in the case reports.

REFERENCES

- ¹ Reid, Mont R.: Studies on Abnormal Arteriovenous Communications, Acquired and Congenital. *Arch Surg.*, **10**, 601-638; **10**, 996-1009; **11**, 25-42; **11**, 237-253, 1925.
- ² Seeger, S. J.: Congenital Arteriovenous Anastomosis. *Surgery*, **3**, 264, February, 1938.
- ³ Wagoner, G. W., and Livingston, A. E.: Application of the Venturi Meter to Measurement of Blood Flow in Vessels. *J. Pharm. & Exper. Therap.*, **32**, 171, 1928.
- ⁴ Robb, G. P., and Weiss, S.: A Method for the Measurement of the Velocity of the Pulmonary and Peripheral Venous Blood Flow in Man. *Am. Heart Jour.*, **8**, 650, 1933.
- ⁵ Holman, Emile: *Arteriovenous Aneurysm*. The Macmillan Company, New York, 11, 1937.
- ⁶ Ney, E.: Über die Bedeutung der Venen bei arterio-venösen Aneurysmen. *Arch. f. klin. Chir.*, **100**, 531, 1912-1913.
- ⁷ Lewis, Thomas, and Drury, A. N.: Obstructions Relating to Arteriovenous Aneurysm. Part II. The Immediate Effects of an Arteriovenous Anastomosis on the Dog's Circulation. *Heart*, **10**, 373, 1923.
- ⁸ Keith, N. M., Rowntree, L. G., and Geraghty, J. I.: A Method for the Determination of Plasma and Blood Volume. *Arch. Int. Med.*, **16**, 547, 1915.
- ⁹ Halsted, W. S.: Congenital Arteriovenous and Lymphatico-venous Fistulae. *Proc. Nat. Acad. Sci.*, **5**, 76, March, 1919.
- ¹⁰ Matas, Rudolph: Diseases of the Vascular System. *Keen's Surgery*, **5**, Chapt. 70, 17, 1909.
- ¹¹ Gibson, J. G., 2nd, and Evans, Wm. A., Jr.: Clinical Studies of the Blood Volume. III. Changes in Blood Volume, Venous Pressure and Blood Velocity Rate in Chronic Congestive Heart Failure. *J. Clin. Invest.*, **16**, 851, 1937.
- ¹² Bird, C. E.: Spontaneous Closure of Arteriovenous Fistulas. *Surgery*, **2**, 924, December, 1937.
- ¹³ Mason, J. M.: Extreme Cardiac Decompensation Following Traumatic Arteriovenous Fistula of the Left Subclavian Vessels. *Am. Jour. Surg.*, **20**, 451-473, May, 1933; *Trans. Southern Surg. Assn.*, **45**, 282-304, 1932.
- ¹⁴ Stone, Harvey B.: Discussion of Mont R. Reid's paper: The Effect of Arteriovenous Aneurysm upon the Heart; with the Report of a Case Studied by Professor R. Matas, Dr. G. R. Herrmann and the Author. *ANNALS OF SURGERY*, **95**, 578-589, April, 1932; *Trans. Southern Surg. Assn.*, **44**, 356-371, 1931.
- ¹⁵ Porter, William B.: The Significance of Cardiac Enlargement caused by Arteriovenous Fistula. *Trans. of the Association of American Physicians*, **52**, 41-48, 1937.

DISCUSSION.—DR. JAMES M. MASON (Birmingham, Ala.) stated that: Though first described by William Hunter, in 1757, surgery had added but little to the treatment of arteriovenous aneurysms until the period of the World War. Osler vigorously opposed operation, and the outstanding contributions of American surgeons up to this time were the recognition and description by Branham, in 1890, of the bradycardic phenomenon which now bears his name, and the report by Matas, in 1902, of the successful operation upon a subclavian arteriovenous aneurysm, the sixth operation which had ever been undertaken for the relief of fistulae involving these vessels.

The number of cases resulting from war wounds was enormous, and became the subject of critical study, which led Gundermann, Caro, Makins, Cazamian and others, between 1915 and 1917, to suspect, and finally to recognize, a definite relationship between arteriovenous communications and associated heart lesions. In 1914, Reid began his experimental work on vascular surgery under the direction of Halsted and stated that: "In the course of two or three years we were fully convinced that a fistula between the large vessels of the neck or legs may cause marked hypertrophy and dilatation of the heart, and, in some instances, cardiac decompensation and death."

The clinical and experimental studies which were undertaken to establish this relationship have been among the most extensive and interesting in the history of medicine. Not until they proved the suspected relationship to be an established fact was the seriousness of arteriovenous aneurysms fully realized. Halsted's death, in 1922, prevented him from taking an active part in the further development of this type of vascular surgery, but the work initiated by him, and continued by his coworkers, has aided immeasurably.

The "Indications for Operation," so clearly stated by Reid in former papers, and the operative procedures which he then advocated, have withstood the most critical tests and are restated in his present paper. To these operative procedures he has added two ingenious methods of closing fistulae difficult of access or too extensive for excision, namely, twisting of the vein and fixation at the site of the fistula; and occlusion of the fistula by transfixion sutures after ligation of the vein and ligation of the artery.

Two questions in regard to arteriovenous aneurysms interest me greatly: (1) Why, in some instances, do cardiac symptoms appear early and progress rapidly, while in others their appearance is delayed and their progress more gradual? (2) What measures may we safely employ when decompensation makes early operation imperative?

In my series of traumatic fistulae are one chronic and four acute cases involving the subclavian vessels. The chronic case was seen three years after his injury. The heart was seriously damaged, but he was at work as a chauffeur and refused operation. He is still at work, nearly eight years after his injury. In one of the acute cases, cardiac decompensation had reached a grave stage at the end of 30 days, and operation was carried out on the seventy-seventh day. In another instance, decompensation resulted in death on the fourth day before any operative measures were undertaken. In two cases operated upon at the end of four months, neither patient showed any heart symptoms of moment.

In the cases of early decompensation, little gross change in the vessels adjacent to the fistula was noted at operation or autopsy. The proximal veins were unobstructed, and, in one instance, seemed rather dilated. The cases without decompensation showed very large varicosities at the site of the fistula and immediately distal to it. In one instance, thrombophlebitis had developed soon after the injury and extended well down the arm. This, however, had subsided and no thrombus was present at the time of operation.

In the experimental fistulae of Holman and Stultz, quoted by Tixier and Arnulf, some cases failed to develop cardiac lesions. They attributed this to thrombosis in the vein proximal to the fistula, which prevented the rapid return of blood to the heart. Reid, Stone, and Holman have made the clinical observation that cardiac symptoms were much improved by proximal ligation of the vein. Tixier and Arnulf, and also Matas, have commented favorably upon the possibility of temporarily relieving early acute decompensation by proximal ligation of the vein; to be followed later, of course, by curative operation upon the fistula itself. The thrombophlebitis which was present in one of my cases may have extended into the proximal vein. The striking similarity in the varices in the other patient suggests the possibility of an unrecognized thrombophlebitis in that case.

In cases under my care there have been two spontaneous recoveries, one in the femoral vessels at the groin and one in the posterior tibial vessels just above the ankle.

DR. JOHNSON MCGUIRE (closing): I think the questions which Doctor Mason has asked are particularly interesting, especially those relative to the

cause for cardiac decompensation in arteriovenous aneurysms and the decision as to the proper time to operate.

In one of the patients Doctor Reid has described, digitalis failed to improve the patient's clinical condition. In this case, progressive increase in the level of the venous pressure occurred. Consequently, operation was decided upon as an emergency procedure. Immediate improvement occurred.

As to why the hearts of certain of these patients become seriously damaged and others do not, we have felt that the size of the fistula is an important factor. With large fistulae, the heart has to pump a large volume of blood and is relatively quickly exhausted by excessive work. Also the fundamental condition of the heart muscle may be of significance, as a patient with arteriosclerotic heart disease would develop failure more rapidly than would a normal individual with fistulae of equal size.

The work of the heart may roughly be calculated if one multiplies the blood pressure by the cardiac output. It is our feeling that constant work, day and night, increases the amount of blood that the heart has to handle, as shown by the measurements with the Venturi meter, and is the probable cause of cardiac failure.

The theory of Sir Thomas Lewis, that heart failure in arteriovenous fistulae is caused by inadequate coronary blood flow, due to the fact that the diastolic aortic pressure is lowered, has been recently challenged, since measurements of coronary blood flow in the presence of artificial arteriovenous aneurysms have failed to demonstrate a significant decline of blood flow in the coronary arteries.

SPONTANEOUS ARTERIOVENOUS FISTULA BETWEEN THE ABDOMINAL AORTA AND THE INFERIOR VENA CAVA

CASE REPORT

PRESENTED IN DISCUSSION OF THE PAPER BY DOCTORS REID AND MCGUIRE
ON ARTERIOVENOUS ANEURYSMS

EDWIN P. LEHMAN, M.D.

UNIVERSITY, VA.

FROM THE DEPARTMENT OF SURGERY AND GYNECOLOGY AND THE UNIVERSITY HOSPITAL, UNIVERSITY OF VIRGINIA SCHOOL OF MEDICINE, UNIVERSITY, VA.

ABNORMAL arteriovenous communications are properly divided into two groups—the congenital and the acquired. The latter group is subdivided into those of traumatic origin, which represent, by far, the largest group of arteriovenous fistulae, and those of so-called spontaneous origin. The latter are the result of disease of the arterial wall; they are caused, in almost all instances, by syphilitic aneurysms that have perforated into contiguous veins. These lesions represent the rarest type of arteriovenous fistula. Although a few cases of spontaneous acquired lesions in the peripheral vessels are on record, it is probable that they have been confused at times with congenital fistulae. Most of the true spontaneous fistulae are complications of aortic aneurysm and occur within the thorax, where perforation may take place into the superior vena cava, the pulmonary vessels or the heart itself. Up until 1930, there had been recorded about 75 instances of spontaneous communication between the aorta and the superior vena cava,^{1, 2, 3, 4, 5} and a somewhat characteristic clinical picture has been described.

Spontaneous fistulae below the diaphragm are apparently very unusual. Matas,⁶ in 1909, stated that Boinet, in collecting 114 spontaneous arteriovenous fistulae, had found only 20 that involved the abdominal aorta and the inferior vena cava. Reid,⁷ in 1925, added no other references, and a complete search of titles, since 1925, revealed no further reports. It is possible, of course, that further examples may be hidden in the rich literature of arteriovenous fistulae, which has not been exhaustively searched. The relative rarity of the abdominal lesion as compared to the thoracic lesion is, of course, explained by the relative infrequency of abdominal aneurysm as compared to thoracic aneurysm.

The appended history of a case of spontaneous arteriovenous fistula connecting the abdominal aorta and the inferior vena cava is reported.

Case Report.—University of Virginia Hospital No. 114,836: J. A., colored, male, age 37, entered the hospital February 1, 1935. The patient had been married for 15 years; his wife had never been pregnant. The past history was unimportant, except for the occurrence of a chancre about ten or 12 years previously. At that time he had been treated with four injections in the arm. In childhood he had received severe barbed wire cuts over the left abdomen and chest.

He had been in good health until 18 months before entrance. At that time he began

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to have pain in the lower part of his back, with swelling of his legs. He was given magnesium sulphate for his edema and several injections in the arm. He improved somewhat until six months before entrance, when both the swelling and the pain became worse. Two months later, he noticed enlargement of the veins of his abdomen and chest, and, two weeks before entrance, a "knocking" over his right abdomen. There was no dyspnea, orthopnea, hemoptysis, or cough. There had been no disturbances in the neuromuscular mechanism, and there was no history of recent injury.

FIG. 1.



FIG. 2.



FIG. 1.—Photograph of the patient showing the swelling of the legs and scrotum, dermatitis of left shin, enlarged superficial veins, scars of barbed wire cuts on abdomen and chest, and the outline of the abdominal masses.

FIG. 2.—Photograph showing the enlarged left thoraco-epigastric vein and scars of the barbed wire cuts.

Physical Examination revealed a rather undernourished Negro with marked swelling of the legs that hampered his walking (Fig. 1). In the left upper abdomen, parallel to the costal margin, was a long keloid, representing the barbed wire cut of childhood. A similar scar crossed the right upper chest. The veins in the upper thighs, the epigastric veins and the anterior thoracic veins were markedly dilated and tortuous (Fig. 2). The veins over the scrotum were enlarged and a marked degree of varicocele was present.

A diffuse apex impulse was seen and felt most prominently in the fifth interspace about 3 cm. lateral to the midclavicular line. A loud systolic murmur was heard all over the precordium. No definite diastolic murmur was present, but the second sound was accentuated. The murmur was transmitted to the neck and axilla. The rhythm

was regular and the rate ranged between 80 and 90. The systolic blood pressure in the arms averaged 160 and the diastolic 70, the average pulse pressure, therefore, being about 90 Mm. Hg.

Abdominal palpation revealed two masses. To the right and just above the umbilicus (Fig. 1) there was felt a mass about 8 cm. in diameter, spherical in outline, firmly fixed and not tender. It presented a marked expansile pulsation with a continuous coarse thrill, accentuated during systole. On auscultation, a loud continuous bruit with systolic accentuation, the typical "machinery murmur," was heard. The same type of murmur could be heard with less intensity over the dilated veins of the abdomen and even over those in the scrotum.

The second mass occupied the position of the lower half of the rectus muscles (Fig. 1). This mass was firm, sharply defined, without pulsation, thrill or bruit, and without tenderness. It remained easily palpable when the rectus muscles were contracted and gave the impression of being superficial.

No other masses were felt, nor was the liver or spleen palpable. There were no signs of free fluid in the abdominal cavity.

The left leg was more swollen than the right (Fig. 1). About the ankles and feet there was pitting edema, but in the calves and thighs the tissues were of brawny consistency and somewhat tender, presenting the typical characteristics of elephantiasis. The circumference of the right thigh was 57 cm.; of the left thigh, 62.5 cm.; of the right calf, 39.5 cm.; and of the left calf, 33.5 cm. Over the anterior portion of the left lower leg was an area of scaly dermatitis. There was slight edema of the penis and scrotum.

The edema of the left leg was too marked to permit palpation of the arterial pulsations. The dorsalis pedis and the posterior tibial arteries were easily palpable on the right and the pulsations seemed of good quality. The systolic blood pressure in the right leg was about 20 Mm. Hg. lower than the pressure in the arms.

Laboratory Data and Special Examinations.—The blood Wassermann and Kahn reactions were strongly positive. The red cell count was 3,440,000, and the hemoglobin 74 per cent (Dare). The white cell count was 8,200 with normal cell distribution. The urine presented occasional white blood cells and rare red blood cells. Phenolsulphonphthalein output was 70 per cent in two hours after intravenous injection. The blood from the enlarged left thoraco-epigastric vein showed an oxygen saturation of 55 per cent as compared with 23 per cent in the blood from the right basilic vein. Although these low readings are somewhat questionable as absolute values, yet, having been measured with the same apparatus and reagents at the same time, they are probably comparable. The venous pressure in the left thoraco-epigastric vein was 400 Mm. of water; that in the left basilic vein 60 Mm. of water. The P-R interval was 0.21 of a second, interpreted as being suggestive of heart disease.

The seven-foot roentgenogram of the chest showed the heart to be somewhat enlarged to the left, with possibly slight enlargement to the right. No abnormality of the thoracic aorta was seen. The width of the heart shadow was 58 per cent of the width of the bony thorax. Roentgenograms of the lungs and abdomen presented nothing remarkable. The lateral view of the lumbar spine revealed no evidence of bone erosion. A pyelogram, after injection of hippuran, revealed no abnormalities, except for slight displacement to the right of the right ureter at the level of the third lumbar vertebra.

Preoperative Diagnosis.—A diagnosis was made of an arteriovenous fistula within the abdomen, involving the inferior vena cava and probably the descending aorta below the exit of the renal arteries. It was believed that the lesion was of spontaneous nature and was probably due to syphilis. The possibility of a preceding aneurysm seemed strong. The marked increase in symptoms in the preceding two weeks, the definite evidence of cardiac enlargement, and the absence of any other possible mode of attack

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rendered surgical intervention inescapable, although it was realized that the chance of cure was remote.

The patient received daily injections of bismuth for four days before operation. During the last 24 hours the patient was digitalized. Preparations were made for transfusion.

Operation.—February 7, 1935: Under drop-ether anesthesia, a long right rectus incision was made. The subcutaneous tissue below the level of the umbilicus consisted almost entirely of a plexus of enlarged, distended, tortuous veins, the ligation of which was tedious and time consuming. On opening the abdominal cavity, no free fluid was found. Bimanual palpation of the firm mass in the lower abdomen revealed it to be within the abdominal wall and incision into it showed it to consist of a hard edema of the lower half of the rectus abdominis muscles, resembling elephantiasis (Fig. 3). It was considered to represent the same changes in the rectus muscle as were present in the legs.

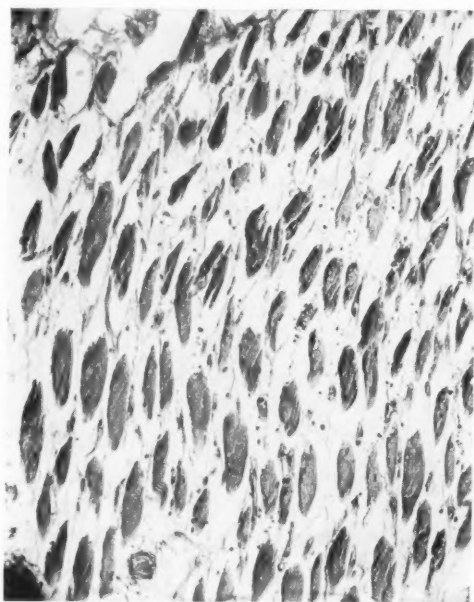


FIG. 3.—Photomicrograph from the rectus abdominis muscle (postmortem specimen) showing edema, fibrosis and cellular infiltration. (Connective tissue stain.)

An incision at the left of the root of the mesentery exposed the pulsating mass which consisted of a typical aneurysm about three inches in diameter. Over it, a marked, coarse thrill could be felt, which was transmitted downward into the region of the inferior vena cava and common iliac veins. The veins in the posterior abdominal wall were markedly dilated, including what was thought to be the spermatic vein.

In the superior wall of the aneurysm, a point of threatening rupture was apparent. It was, therefore, thought best to control the circulation entering the sac before further dissection. All the tissues in the neighborhood of the aneurysm were markedly inflamed and dissection was difficult. Tapes were placed about the abdominal aorta, the vena cava above the sac, and two vessels below the sac, which were considered to be the aorta and the vena cava. When the aorta above the sac was ligated (Chart 1), there was no drop in pulse, but the thrill disappeared and the blood pressure rose about 30

Mm. Hg. During the dissection, the weak point in the sac ruptured and had to be plugged with the finger. After quadruple ligation, blood still escaped freely from the sac, and further dissection was attempted to find other entering vessels. At this time it became obvious that the retroperitoneal tissues, distal to the sac, were thickened, edematous and scarred, presenting the pathologic appearance of elephantiasis, similar to that described in the legs and rectus abdominis muscles. It was impossible to identify any structures, including even the larger vessels. With increasing size in the accidental opening in the sac, the situation became critical. It was necessary to pack the sac with gauze, pressure upon which controlled all bleeding.

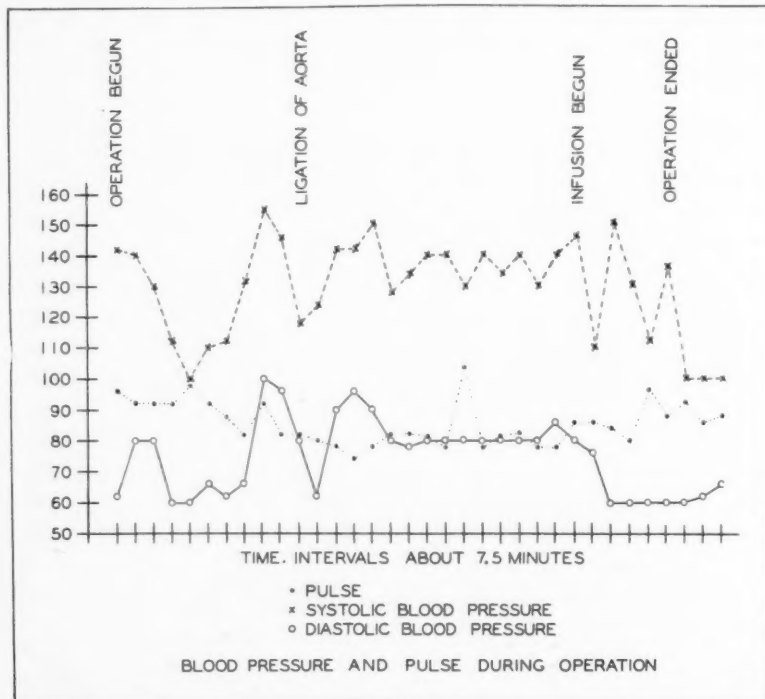


CHART 1.—Showing the ranges of blood pressure and pulse rate during the operation. Note the absence of important changes, particularly in the pulse rate and pulse pressure, after ligation of the abdominal aorta. Note, also, the absence of shock at the end of the operation, which lasted three and one-half hours.

Any attempt to remove the gauze resulted in the free escape of venous blood. The sac was, therefore, repacked and the abdomen was closed with silk. No stay sutures were employed, in order to avoid the possibility of obstructing the deep epigastric arteries as potential or actual collaterals.

During the operation the patient received 1,200 cc. of normal saline solution. In spite of an operation lasting three and one-half hours and a considerable loss of blood, he was returned to the ward in excellent condition, with a pulse of 90 and a blood pressure of 130/60 (Chart 1).

Postoperative Course.—Shortly thereafter, the blood pressure dropped to 100/60, and later numerous extrasystoles developed. At about this time there was a sharp rise in systolic blood pressure to 160, which was not sustained; the blood pressure shortly before death was 120/80. The patient died 15 hours after the end of the operation. After operation the patient voluntarily moved the feet and legs slightly, but during the last

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ten hours of life he was unable to move them. No venous pressures were obtained during the agonal period.

Autopsy.—At postmortem examination, the heart was found definitely enlarged and hypertrophied. It weighed 490 Gm. The walls of both ventricles were somewhat increased in thickness. There was moderate dilatation of the right side of the heart and none of the left. There was no excess pleural fluid, but there was a slight degree of chronic passive congestion of the liver. Except for syphilitic aortitis, there were no other abnormalities related to the presenting lesion.

It was found that the fistula, measuring 1 cm. in diameter, lay at the lower pole of an abdominal aneurysm, which involved the lower portion of the descending aorta from below the spermatic arteries to the bifurcation (Fig. 4). The inferior vena cava was firmly attached to the wall of the aneurysm above the fistula and was markedly narrowed. The appearances suggested that there had been marked compression of the vena cava by the aneurysm preceding the occurrence of the fistula. The inferior mesenteric artery which had not been recognized at operation was found to originate from the posterior portion of the sac wall. The vessels that had been ligated were found to be the descending aorta and the inferior vena cava above the sac, and the two common iliac arteries below the sac. Without control of the inferior mesenteric artery, the lumbar arteries and the vena cava below the sac, the free bleeding after supposed quadrilateral ligation was readily explained.

The reason for the difficulty in identification of vessels also became apparent. On account of the retroperitoneal inflammatory tissue and edema, presumably the result of prolonged venous stasis, the pathologist could identify the vessels only after extensive dissection with the material removed from the abdomen.

In retrospect, it is believed that the patient suffered for about 18 months from compression of the inferior vena cava as the result of an aortic aneurysm. It is probable that perforation of the aneurysm into the vena cava occurred not more than 16 weeks, and possibly only two weeks, before admission. It is felt that such a large fistula, situated such a short distance from the heart, should have caused more marked cardiac symptoms and more apparent signs of cardiac failure unless the duration had been relatively short.

It is interesting that the ligation of the abdominal aorta was not associated with any immediate change in pulse rate, although there was a rise of 30 Mm. Hg. in systolic pressure. It is also interesting that, in spite of arterial control of the fistula, there was no immediate marked diminution of the pulse pressure.

It is probable that the condition was inoperable, although the necessity for an operative attempt was obvious. The difficulty encountered by the pathologist in identifying the vessels under favorable postmortem conditions

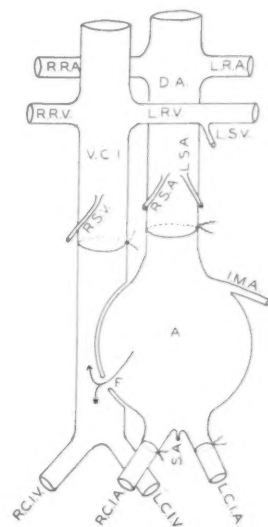


FIG. 4.—Diagram of actual conditions as found at autopsy. The position of the fistula and the compression of the proximal vena cava are indicated. The points of operative ligation are indicated. D. A. = descending aorta; V. C. I. = vena cava inferior; R. R. A., L. R. A. = right and left renal artery; R. R. V., L. R. V. = right and left renal vein; R. S. A., L. S. A., = right and left spermatic artery; R. S. V., L. S. V. = right and left spermatic vein; A. = aneurysm; I. M. A. = inferior mesenteric artery; F. = fistula; S. A. = sacral artery; R. C. I. A., L. C. I. A. = right and left common iliac artery; R. C. I. V., L. C. I. V. = right and left common iliac vein.

was so great, and the vessels that would have had to be identified and ligated for complete control of the fistula were so numerous, that, under relatively unfavorable operative conditions, the task became insuperable.

The cause of death is not obvious. Shock or cardiac failure cannot be assumed in the face of an agonal systolic blood pressure of 120 Mm. Hg.

REFERENCES

- ¹ Pepper, W., and Griffith, J. P. C.: Varicose Aneurysms of the Aorta and Superior Vena Cava. *Am. J. Med. Sci.*, **100**, 329, 1890.
- ² Humphry, L.: Aneurysm of the Aorta Communicating with the Superior Vena Cava. *Brit. Med. J.*, **2**, 1046, 1910.
- ³ Shennan, J.: Spontaneous Arteriovenous Aneurysm in Thorax. *Edinburgh Med. Jour.*, **32**, 325, 1925. (Cited by House and Goodpasture.)
- ⁴ House, S. J., and Goodpasture, E. W.: Spontaneous Arteriovenous Aneurysm in the Thorax. *Am. Heart Jour.*, **3**, 682, 1927-1928.
- ⁵ Packard, M., and Wechsler, H. F.: Arteriovenous Aneurysm between Aorta and Superior Vena Cava. Case Report. *Am. Heart Jour.*, **6**, 281, 1930-1931.
- ⁶ Matas, R.: *Keen's Surgery*, **V**, 290 ff., 1909.
- ⁷ Reid, M. R.: Abnormal Arteriovenous Communications, Acquired and Congenital. *Arch. Surg.*, **10**, 996, 1925.

CONGENITAL ARTERIOVENOUS FISTULA OR FISTULAE

CASE REPORT

PRESENTED, BY TITLE, IN DISCUSSION OF THE PAPER BY DOCTORS REID AND MCGUIRE ON ARTERIOVENOUS ANEURYSMS

WALTER D. WISE, M.D., AND EPHRAIM T. LISANSKY, M.D.

BALTIMORE, MD.

Case Report.—J. D., white, male, age 33, was admitted to the Mercy Hospital, Baltimore, July 4, 1937. He complained of venous enlargements which extended from the left nipple line to the terminal point of the left foot and covered a corresponding area on the posterior surface. These varicosities were present on the left side only. He stated that the condition had existed since birth, and had neither diminished nor increased in severity or anatomic extent. He also complained of a slight limp, and occasional pain in the region of the varicosities. He has never at any time been incapacitated due to this condition. He had never had any serious illness in his life, other than the complaint mentioned. His family history was entirely negative.

Physical Examination showed a young man, lying comfortably in bed with no apparent discomfort. *Head:* Scalp and ears negative; *eyes*—very nearsighted, both optic nerves normal, retinae normal, congenital myopia of seven to eight diopters in each eye; *nose*—breathing space rather limited and septum deviated to the right; *mouth*—teeth in rather good condition, two artificial teeth in lower jaw, all else negative, mucous membranes of good color. *Neck:* No abnormal pulsations, no enlargement of vessels, a few small lymph nodes palpable on left side. *Chest:* Lungs negative. *Heart:* Apex beat was not visible or palpable. No abnormal thrusts, thrills or shocks. Percussion outline was clearly normal and apex within midclavicular line in fifth interspace. Auscultation—mitral sounds slightly muffled but no murmurs present. Aortic area negative. Pulmonic area negative. Pulse of equal force and rate, the latter remaining between 70 and 80 during his entire hospitalization.

General examination showed an enlargement of venous channels, both small and large, which was restricted entirely to the left side of the body. Anteriorly, the varicosities began about one inch above the nipple line and posteriorly at a corresponding level, sweeping around the left side of the chest. The venous enlargements were multiple, bluish colored, discrete and confluent. In the sitting or standing position, they became much more prominent. Anteriorly, they continued down over the left side of the abdomen and presented a number of large single dilations. Posteriorly, there were no varicosities from the inferior angle of the scapula to the level of the crest of the ilium where they began to present a network of anastomosing vessels. The penis and scrotum were also affected, the dilations being limited to the left side. The lower extremity was particularly affected. The left thigh, knee, leg, and especially the ankle, foot and toes on the left side were covered by a conglomerate mass of dilated vessels (Fig. 1). There was no tenderness on palpation. The left side of the patient, from the nipple line down, was noticeably warmer to the touch than was the right side. There was no bruit heard, no thrill felt and no subjective sensation of thrill or throbbing. The skin over the right scapula was covered by a rather heavy growth of hair. This was not present on the opposite side. The upper extremities were not affected by venous enlargement. Blood pressure in both arms was 110/70. Pressure over the left femoral artery caused no change in heart rate. The reflexes in the lower extremities were active and equal. Muscle tone in the left lower extremity was much weaker than in the right. On palpation the muscles of the left lower extremity were much smaller and softer than corresponding

muscles on the opposite side. However, the left lower extremity was larger in circumference at almost every point than the right lower extremity. Five centimeters above the knee, the left thigh was 1.3 cm. larger than the measurement at the corresponding point on the opposite side. The left knee was 2.5 cm. larger than the right knee. Over the midpoint of the belly of gastrocnemius, the left side was 2.5 cm. larger than the measurement at the corresponding point on the opposite side. The ankle on the left side was 5 cm. larger than the right. The toes on the left foot showed hypertrophy of the cutaneous tissue, and each toe measured more in circumference than the corresponding toe on the opposite foot. The left lower extremity was 3.5 cm. longer than the unaffected extremity. With the patient in the erect position, the left leg, ankle and foot became swollen and cyanotic and the vessels became markedly engorged.



FIG. 1.—Showing varicosities from above the left nipple line to the toes. (Infra-red technic.) Insert: Same as Fig. 1, with ordinary photography.

directly into the bellies of the gastrocnemii. This thermocouple was originally designed for the measurement of internal temperatures of animals. It was used in conjunction with a sensitive galvanometer. The calibration of the thermocouple galvanometer combination was performed by immersing the two junctions in two beakers of water at different temperatures, these temperatures being read by thermometers. The sensitivity of the combination was found to be 7.0 Mm. of deflection = 1 degree C., and 3.9 Mm. of deflection = 1 degree F. Therefore, a difference in temperature could be measured to within about 1/70 of a degree C. or 1/40 of a degree F.

Roentgenologic Findings.—Left foot—swelling of soft tissues on dorsal and inner surface of foot. Bone atrophy with striation of both metatarsal and tarsal bones. No bone destruction or new bone formation. Left ilium—negative. Left femur and knee—marked striation. Left leg—marked forward and inward bowing of tibia with striation and numerous small areas of bone absorption. Bone not enlarged. Left fibula—enlargement with bending forward and inward, and rather marked striation with numerous areas of bone absorption throughout (Fig. 2). Right tibia, fibula (Fig. 3), femur, hip and ilium—no pathology shown.

* Two indirect methods used: (1) Gaertner's; and (2) modified Eyster's. Venous pressure was taken at corresponding points on each side with lower extremities in similar position.

Blood pressure variation in lower extremities:

Arterial: Right popliteal region 130/78.
Left popliteal region 156/100.

Venous:* the venous pressure was 40 Mm. of water higher on the left side than on the right side.

Temperature Variations:

(1) Water bath immersion method: 2° F. difference. The left foot consistently revealed a higher temperature than the right foot.

(2) Thermocouple determinations: Cutaneous, subcutaneous, and intramuscular temperature determinations of corresponding points on both sides showed that the left side registered a consistently higher temperature (Table I).

These temperature differences were measured by means of a copper constantan thermocouple, the junctions of which were mounted inside hypodermic needles. Temperature readings were obtained by placing the needles on the skin, subcutaneously and

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TABLE I

Region of Body	Inc. Temp. on Left*
I. Ankle (surface).....	2.1° F.†
II. Middle third of leg (surface)	
A. Particularly venous region.....	2.3° F.
B. Region devoid of abnormal veins.....	0.4° F.
III. Intramuscular determination (gastrocnemius muscle)—1 inch below skin..	1.2° F.
IV. Thigh (subcutaneous).....	0.5° F.
V. Upper popliteal region (surface).....	1.2° F.
VI. Posterior iliac region (subcutaneous).....	1.1° F.
VII. Midaxillary region (surface).....	0.4° F.

* The degree of temperature difference between the right and left sides, or to what extent the left lower extremity and left side of chest and abdomen were warmer than corresponding points on the right side.

† Temperature accuracy was to 1/70 of a degree centigrade or 1/40 of a degree Fahrenheit.

FIG. 2.



FIG. 3.



FIG. 2.—Left or affected tibia and fibula. See text for description.

FIG. 3.—Right or unaffected tibia and fibula.

Electrocardiographic Findings.—Essentially normal tracing. No evidence of any pathologic condition being present.

Oxygen Determinations in Venous Blood of Right and Left Lower Extremities:

(a) Blood taken at corresponding levels on both sides from venous channels.

Right or Normal Side	Left or Affected Side
3.79 vol. % or 49.9% O ₂ saturation	7.59 vol. % or 100% O ₂ saturation
9.4 vol. % or 39.6% O ₂ saturation	23.7 vol. % or 100% O ₂ saturation
11.85 vol. % or 47.5% O ₂ saturation	24.88 vol. % or 100% O ₂ saturation

Therefore, the venous blood of the left lower extremity which is the one affected, has a higher O₂ concentration than the venous blood of the normal limb.

(b) Venous blood on right side—bluish or dark red. Venous blood on left side—bright red.

Summary.—Points substantiating a diagnosis of congenital arteriovenous fistulae or fistulae:

- (1) Visible venous enlargements on left side since birth.
- (2) Increased venous pressure on left side.
- (3) Venous blood of much brighter hue on left side than venous blood on right side.
- (4) Higher O₂ saturation of venous blood on left side than O₂ saturation of venous blood on right side.
- (5) Left lower extremity longer than right lower extremity. Left lower extremity larger in circumference than right lower extremity at corresponding points.
- (6) Temperature of left lower extremity approximately 2° F. higher than right lower extremity.
- (7) Left lower extremity shows some evidence of muscular atrophy.
- (8) Bone atrophy, bowing, striation and absorption on left side. Slight degree of bone enlargement.

REFERENCES

- ¹ Samuels, Saul S.: *The Diagnosis and Treatment of Diseases of the Peripheral Arteries.* Oxford Medical Publications, 157, 1936.
- ² Horton, B. T., and Ghormley, R. K.: Congenital Arteriovenous Fistula. *Proc. Staff Meet. Mayo Clinic*, **8**, 773, 1933.
- ³ Horton, B. T.: Arteriovenous Fistula Involving the Common Femoral Artery Identified by Arteriography. *Proc. Staff Meet. Mayo Clinic*, **8**, 189, 1933.
- ⁴ Lewis, D.: Congenital Arteriovenous Fistulae. *Lancet*, **2**, 621, 1930.
- ⁵ Horton, B. T., and Dry, T. J.: Traumatic Arteriovenous Fistula Involving the Right Femoral Artery and Vein. *Arch. Surg.*, **33**, 248-252, 1926.
- ⁶ Christopher: *Textbook of Surgery.* Saunders, 149, 1936.
- ⁷ Reid, Mont R.: Studies of Abnormal Arteriovenous Communications—Acquired and Congenital. 1—Report of a Series of Cases. *Arch. Surg.*, **10**, 601, 1925. *Idem*: 2—The Origin and Nature of Arteriovenous Communications, Cirroid Aneurysms and Simple Angiomas. *Arch. Surg.*, **10**, 996, 1925. *Idem*: 3—The Effects of Abnormal Arteriovenous Communications on the Heart, Blood Vessels and Other Structures. *Arch. Surg.*, **11**, 25, 1925.
- ⁸ Pemberton, J. J., and Saint, J. H.: Congenital Arteriovenous Communications. *Surg., Gynec. and Obstet.*, **46**, 470, 1928.
- ⁹ Pemberton, J. J.: Arteriovenous Aneurysms. *Arch. Surg.*, **16**, 469, 1928.
- ¹⁰ Holman, Emile: Experimental Studies in Arteriovenous Fistulae. *Arch. Surg.*, **9**, 822, 1924.
- ¹¹ Holman, Emile: The Physiology of an Arteriovenous Fistula. *Arch. Surg.*, **8**, 64, 1923.
- ¹² Callander, C. L.: Study of Arteriovenous Fistula with an Analysis of 447 Cases. *Johns Hopkins Hosp. Rep.*, **19**, 259, 1920.
- ¹³ Horton, B. T.: Hemihypertrophy of Extremities Associated with Congenital Arteriovenous Fistula. *J.A.M.A.*, **98**, 373, 1932.
- ¹⁴ De Takats, G.: Vascular Anomalies of the Extremities. *Surg., Gynec. and Obstet.*, **55**, 227, 1932.
- ¹⁵ Horton, B. T., and Smith, F. L.: Sclerosing Treatment of Congenital Arteriovenous Fistulae, Report of 2 cases. *Proc. Staff Meet. Mayo Clinic*, **12**, 17, 1937.
- ¹⁶ Holman, Emile: *Arteriovenous Aneurysm.* The Macmillan Company, N. Y., 1937.

HEMINEPHRECTOMY IN DISEASE OF THE DOUBLE KIDNEY

REPORT OF FOURTEEN CASES

EDWIN BEER, M.D., AND WILLIAM H. MENCHER, M.D.

NEW YORK, N. Y.

IN THIS paper, we wish to present an analysis of 104 cases of double kidney, and, at the same time, to discuss the operation of heminephrectomy as a conservative measure in dealing with certain types of disease with this anomaly. We shall be able to illustrate this operative procedure by submitting a series of 14 cases in which heminephrectomy was the operation of choice.

The frequency of double kidney with complete or incomplete duplication of the ureters varies, according to different authors, from 1.2 to 10 per cent. It is generally stated that a conservative average for the incidence of this anomaly is 3 to 4 per cent of all autopsies. The anomaly may be unilateral or bilateral, with complete double ureters or bifid ureters. In view of the refinements in diagnostic technic which have been developed in recent years, it should be possible to study a case completely, so that therapeutic procedure can be decided upon in advance.

We have been able to collect a total of 104 cases of double kidney from the records of Mount Sinai Hospital. Analyses of this group are shown in the various appended tables.

TABLE I

Total Cases	Unilateral Double Kidney	Bilateral Double Kidney
104	89 (85.5%)	15 (14.5%)

TABLE II

Type of Lesion	Total	Right Side	Left Side	Double Ureter or Complete Duplication	Bifid Ureter or Incomplete Duplication	Symptoms	Showing Pathology	Upper Pole	Lower Pole	Both Poles	Operations
Unilateral double kidney	89	43	46	60 (67%)*	28 (31%)	51	48†	8	27	11	22
Bilateral double kidney	15	0	0	13	2	11	9‡	0	4	2	3

* In one case no information could be obtained as to duplication of ureter.

† In two cases pole not mentioned.

‡ In three cases no information as to the site of the lesion.

Of the 104 cases, there were 89 of unilateral double kidney (85.5 per cent), and 15 cases of bilateral double kidney (14.5 per cent).

Analyzing 89 unilateral double kidneys, 46 were found on the left side and 43 on the right side. Fifty-one cases had symptoms referable to the particular kidney, whereas, the 38 remaining had no symptoms. Sixty cases had complete duplication of the ureter (68 per cent); 28 cases showed incom-

plete duplication (31 per cent), and in one case information concerning this point could not be obtained. Forty-eight cases showed definite lesions (Table II). In 41 cases no definite lesion was discernible, the findings of duplication being made either at postmortem (24 cases) or clinically during routine examination. It is interesting to note that the kidney on the side opposite to the one which produced the presenting symptoms was found to contain a lesion in 17 instances.

TABLE III

Type of Lesion	Upper Pole	Lower Pole	Both Poles
Unilateral double kidneys	8 cases: Calculus—3 Pyonephrosis—1 Ectopic ureter—2 Ureterocele—1 Hydronephrosis—1	27 cases: Calculus—11 Pyonephrosis—9 Hydronephrosis—4 Tuberculosis—1 Carcinoma—1 Ureteritis cystica—1	11 cases: Pyonephrosis—3 Pyelonephritis—3 Hydronephrosis—4 Infection—1
Bilateral double kidneys	No cases	4 cases: Pyonephrosis—3 Infected hydronephrosis both lower poles—1	2 cases: Pyelonephritis—1 Hydronephrosis—1

Of the 15 cases of bilateral double kidneys, 13 cases (87 per cent) showed complete duplication of the ureter on both sides. In the remaining two cases, the ureters were incompletely duplicated on both sides. There were symptoms in 11 cases. In four instances, the lesion was limited to the lower pole, in two instances both upper and lower poles were involved, and in three cases, no information was given as to the site of the lesion. Three of the cases were operated upon, two having an heminephrectomy and the third a complete nephrectomy. In six cases there was no definite lesion.

TABLE IV

TYPES OF OPERATIONS

Total number in both groups—25 cases:
Heminephrectomy—14 cases
Complete nephrectomy—6 cases
Ureterolithotomy—3 cases
Pyelolithotomy—1 case
Cystoscopic cauterization of ureterocele—1 case

There were 62 patients in both groups who had symptoms. Of these, 25 cases were operated upon (Table IV). In other words, the incidence of symptomatology in all patients with a double kidney in our series was about 60 per cent, and operative therapy was carried out in 25 per cent of all cases, with or without symptoms. Braasch and Scholl¹ reported 30 operations in 144 cases (21 per cent). However, if the patient had symptoms referable to the kidney, the operative rate was 40 per cent. It is also of interest to note that of the 62 patients who had symptoms, 55 of these (90 per cent) showed

HEMINEPHRECTOMY

definite lesions. An analysis of this series corroborates the impression that this type of anomaly predisposes the organ to serious pathologic changes, such as ordinary infection, tuberculosis, tumor formation, calculus and, most frequently, to obstructive conditions, leading to hydronephrosis or pyonephrosis (hydronephrosis or pyonephrosis was the lesion in 27 out of the 52 cases [52 per cent] in which the nature of the lesion was mentioned).

In the present series the predominance of pathology in the lower pole (31 out of 39 cases) is evident. This seems to be somewhat at variance with the incidence in the cases recorded in the literature as to the site of the lesion. It is readily accounted for by the fact that in many of those cases reported in

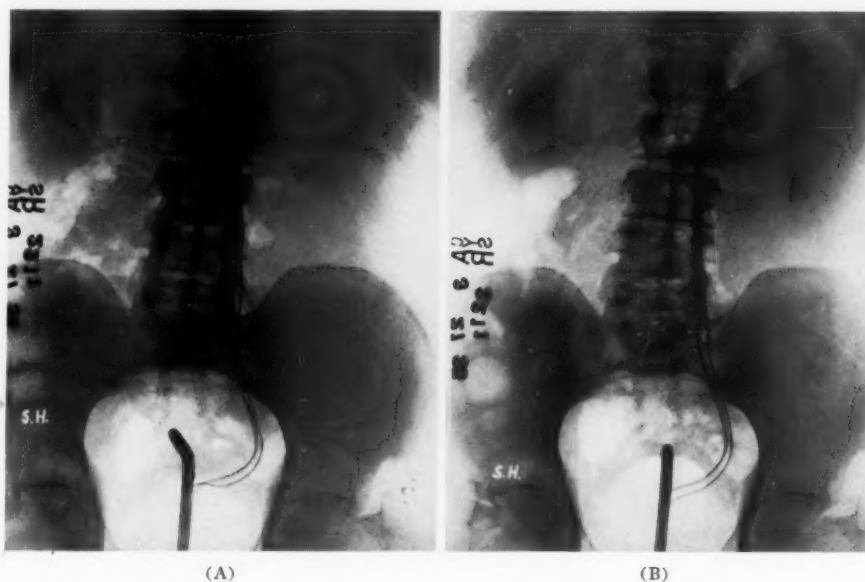


FIG. 1.—Tumor of the lower pole of a double kidney. (A) Retrograde pyelogram of the lower pole. (B) Retrograde pyelogram showing both pelves.

which the upper pole contained the lesion were those of incontinence, in which an ectopic ureter opening was found. As is well known, these ectopic openings* always communicate with the upper pole. In our series, only two cases of ectopic ureter were present. In six of the 25 cases, in which operation was performed, heminephrectomy was contraindicated, and complete nephrectomy was necessary. In one case, a carcinoma was present in the lower pole (Fig. 1A and B). In one case, there was a tuberculous process in the lower

* The one case in which the ectopic ureter communicated with the lower pole, as quoted by Moulonquet,* may have involved a misinterpretation of the operative findings. In Case 14, for example, at the original nephrostomy operation, the mass apparently lay in the region of the lower pole, although subsequent events showed this to have been a huge pyonephrosis of the upper pole which, by sheer weight and size, had so overlapped the lower pole that the impression was gained that we were dealing with lower pole pathology. Since the operative details in M. Bourgeois' case are not recorded, it is best to withhold judgment until definite proof is afforded.

pole. In the remaining four cases, the disease had either involved both poles, or there was little functioning renal tissue in the other half.

Anatomy of Double Kidney.—The two halves of the kidney may show distinct separation, as evidenced by shallow to deep grooving in the paren-



FIG. 2.—Case of complete duplication of kidney and ureters, showing communication between upper and lower pelves. This was proven by injecting blue stained radiopaque solution through one of the two ureter orifices in the bladder and noting blue spurting through the other orifice. At the same time, the above roentgenogram was obtained. Further corroboration was obtained by placing one catheter in the second of the two left-sided orifices and repeating the above procedure. The results obtained were the same. There was no history of previous disease of this kidney.

chyma. The external separation may extend into the interior and the two pelves may be completely separated, either by a band of renal parenchyma or by a fibrous septum. Occasionally no external sign of demarcation between

the upper and lower poles may be noted. Instances have been reported in which wide separation has taken place between the two halves. Some of these fall into the class of true supernumerary kidneys. The existence of true intercommunication between the pelves has been denied by many, but Braasch,² Israel³ and Joseph⁴ have described such cases, and are agreed that true communication may exist. Mann⁵ (Fig. 2) and MacKenzie⁶ have each described this type of anomaly. Gruber,⁷ however, states that such intercommunication is due to fistulization, produced by gradual erosion of parenchyma, usually by stone.

The lower pole is usually the larger of the two halves. Its pelvis and caliceal system may be perfect in outline, whereas, the pelvis and calices in the upper pole are rudimentary and much smaller. Double kidney in one or both halves of a horseshoe kidney may occur, as in Case 4 (Fig. 4) in our series of heminephrectomized patients.

In cases where the ureter is completely duplicated, one should remember Weigert's law, namely, that the laterally placed ureteral orifice leads to the lower pole. The ureter to the upper half, in its uppermost course, may be close to the dilated pelvis of the lower diseased half. It may be displaced out of its normal position, or firmly adherent to the pathologic lower half. One must guard against injury to a ureter so located. Below, the ureters may be enclosed in the same sheath. If a diagnosis of duplication has not been made preoperatively, division of the normal ureter, as well as the one going to the diseased portion of the kidney, might result during the operation, involving the exposure of the lower half of the ureter.

The blood supply to the double kidney determines, in many instances, the feasibility of operative separation of the two halves. In most instances, two distinct sets of vessels supply the respective poles; in other cases the vessels to one pole may arise as branches from the vessels supplying the other pole. The ideal situation, from an operative standpoint, lies in the first anatomic disposition. The other anatomic arrangement contraindicates heminephrectomy.

Diagnosis of Double Kidney.—With the advent of cystoscopy and pyelography by the retrograde and excretory methods, the diagnosis of the double kidney is made a relatively simple matter. The intravenous administration of a dye, such as indigo carmine, is of great aid in recognizing accessory ureteral openings, if their recognition should prove otherwise difficult. Ureteropyelography by either method will be of help in those cases in which the ureters join before they reach the bladder. Suspicion of a double kidney should always be aroused when a small, incomplete pelvis, with minimal number of calices in the upper or lower portion of a kidney, is noted. What appears to be a posed kidney, without ureteral kinking on the same side, might be indicative of a lower pole of a double organ. Likewise, a pelvis, occasionally rudimentary and placed abnormally high under the ribs, might be indicative of the upper pole of a double kidney. The presence of an ectopic ureteral opening assures the investigator that he is probably dealing with a double

kidney and that the ectopic ureter leads to the upper pole. Patients suffering from such an anomaly are usually incontinent and can be cured of their incontinence by heminephrectomy, if the other half of the kidney is normal.

Heminephrectomy.—There has been a tendency in renal surgery toward conservation of as much kidney parenchyma as possible. "Partial nephrectomy" or "partial resection" of the single kidney for localized hydro- or pyocalix, with suture of the remaining cut renal parenchyma, is an example of such an attempt at conservatism. The ideal situation for such procedure presents itself, of course, in the double kidney, in which a separate blood supply and pelvis exist for each of the two poles, as well as a partially or completely duplicated ureter. We shall employ the term "heminephrectomy," restricting its use to describe the operation in which a portion of the double kidney containing a separate pelvis is removed, after ligation of its separate blood supply and severance of its respective ureter. The terms "partial nephrectomy" or "partial resection" of the kidney should be reserved for procedures on the unduplicated organ. We exclude in this series those cases in which one-half of a horseshoe kidney has been resected.

Ionel⁹ collected, in the literature up to 1935, 52 cases in which heminephrectomy was performed. To these he added four of his own, in which the disease was pyelonephritis located in the upper pole. We have been able to collect 30 additional cases in the literature^{10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 32} up to 1938, bringing the total up to 86 cases reported. To these we wish to add 14 of our own cases from the records of Mount Sinai Hospital, making a total of 100 cases in the literature at the present time.

An analysis of these 100 cases shows that in 51 instances the disease for which the operation was performed was in the upper pole, 37 in the lower pole, and in 12 no mention was made as to the site of the lesion. In the 51 upper pole cases are included a fair number of instances in which an ectopic ureter opening was found. Almost invariably, this ureter leads to the upper pole.

TABLE V
TOTAL COLLECTED CASES OF HEMINEPHRECTOMY

Period	Total	Upper Pole	Lower Pole	No Information
Cases up to 1935.....	56	26	25	5
Cases 1935-1938.....	30	21	2	7
Present series.....	14	4	10	0
Total.....	100	51	37	12

Indications and Contraindications for Heminephrectomy.—It can be stated that heminephrectomy may be performed upon double kidneys in which one segment is involved in a hydro- or pyonephrotic, calculous or infectious process, providing one of the following conditions or situations is not present:

(1) Tuberculosis. Here the tuberculous process very frequently involves the parenchyma of the other pole. However, Legueu²³ and Heymann²⁴ have each reported one case in which heminephrectomy was performed upon a

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double, solitary kidney with good results. We might modify the indication in these cases by stating that where the kidney is solitary and there is no involvement of the other half, the procedure is justified.

(2) Malignant tumors are contraindications to this procedure. In the series, we referred elsewhere to a case in which there was a tumor in the lower pole.

(3) Nonfunction of the remaining portion of the kidney.

(4) Single blood supply to both poles.

(5) Technical difficulty in separating the two halves of the kidney.

(6) Advanced disease of both halves (mild infection in one-half is not a contraindication) and especially hematogenous infection.

(7) Communication between the two pelves.

Operative Technic.—The various points in the technic of heminephrectomy, as practiced on our service, may be summarized as follows:

(1) Exposure of the kidney through a loin incision. Occasionally, where the upper pole is the one to be removed, and the kidney is situated at a high level, resection of the twelfth rib may be necessary. The extent of mobilization of the kidney should be minimal, because of the danger of rupture of aberrant vessels necessary for the nourishment of the remaining part of the kidney.

(2) Exposure of the ureters and tracing of their course to the respective poles.

(3) Exposure of the blood supply to both halves and notation of its distribution. The operation will depend a great deal for its success upon whether or not the blood supply to the remaining half of the kidney is adequate.

(4) Location of a line of separation of the two halves.

(5) Ligation of the respective ureter. It is to be remembered that the two ureters very often lie in the same sheath. Separation of the two ureters, without injury to the one going to the remaining half, is essential. If the ureter is normal, the point of ligation should be as low as possible. In those cases, however, in which the ureter is markedly diseased and dilated, complete ureterectomy is preferable. The lowermost portion of the ureter may be isolated and divided close to the bladder by continuing the original lumbar incision, or by making a separate incision in the lower quadrant of the abdomen, identifying the ureter by pulling gently on the freed ureter above. Where the ureter is ligated at the bladder, it is essential to prevent "blowout" of the tied stump postoperatively, by keeping the bladder relatively empty for a number of days by employing an indwelling catheter or by catheterization at regular intervals. Division of the ureter is accomplished by the carbolized knife. In those cases in which there has been a separate incision for juxtavesical division of the ureter, it is best to prevent contamination by covering the proximal stump with a finger cot before pulling the ureter along the tract leading to the lumbar incision. In the case of the "Y" ureter, the ligation should be made a short distance above the junction of the two ureters. The reason is obvious.

(6) Ligation of the blood supply to the portion of the kidney to be removed may cause a blanching of the parenchyma, which will aid in locating the zone of demarcation between the upper and lower poles. Delineation of a separation zone is aided in many cases by inserting a finger into the diseased pelvis and using this as a guide in dividing the upper from the lower pole.

(7) After removal of the diseased portion, mattress sutures, underpinned with fat, muscle or fascia, according to the Beer technic,²⁵ may be employed to secure hemostasis and at the same time to suture the cut ends of the remaining tissue.

(8) Rubber dam drain and closure of the wound.

Variations in Technic.—Von Lichtenberg²⁶ decapsulates the diseased portion after ligating the vessels. He notes the blanching in the portion deprived of its blood supply before making the separating incision. This obviates compression of the vessels to the healthy portion, as practiced by some. The decapsulated flaps are inserted into the wedge shaped cavity at the point of resection. Lennander,²⁷ Wright²⁸ and others perform nephropexy of the remaining half (usually lower pole). Lowsley²⁹ employs ribbon-gut for approximation of the cut edges of renal parenchyma. Miller³⁰ and Hicks¹⁸ both ligated the ureter to the diseased portion of the kidney in an attempt to cause atrophy, since, in the two cases reported, heminephrectomy was not feasible. In Miller's case the upper pole of the right kidney was diseased, and at operation only one renal vascular pedicle was present. He resected 5 cm. of the upper ureter and ligated the stump. The patient made an uneventful recovery. In Hicks' case, there was a hydronephrosis in the upper half. The patient's chief symptom was pain. The ureter to the upper half was ligated, in the hope that atrophy would occur, but there was no relief from the pain. At a second operation, an heminephrectomy was performed.

CASE REPORTS

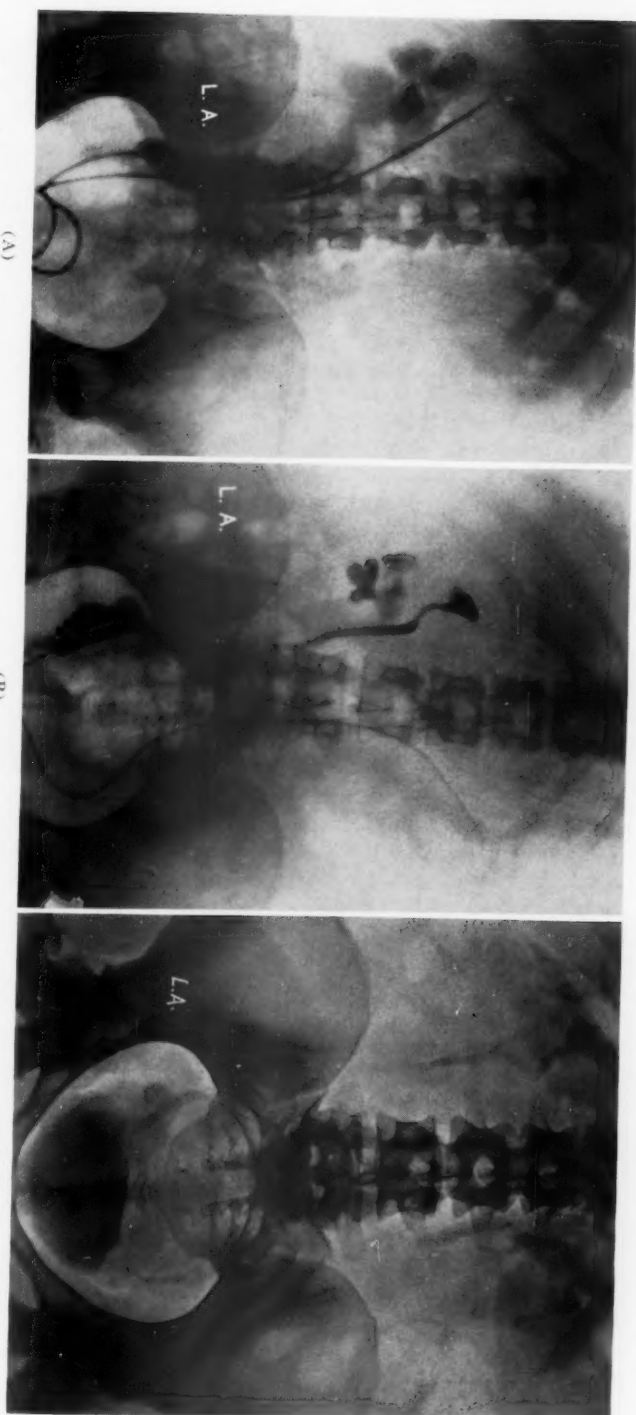
Case 1.—L. A., female, age 29. *Diagnosis:* (1) Complete duplication right kidney and ureter. (2) Pyonephrosis lower half. *Operation:* Right heminephrectomy and ureterectomy. *Result:* Well.

The patient gave a history, extending back six years, of pain in the right kidney area, associated with frequency, pyuria, hematuria and dysuria. Cystoscopic and pyelographic studies revealed a single, normal kidney on the left side, a completely duplicated kidney and ureter on the right side, and a pyonephrosis of the lower half of the double kidney. The upper pole of the double kidney was normal (Fig. 3A and B).

Operation.—July 19, 1932: Heminephrectomy, lower half, and complete ureterectomy (E. Beer). The right kidney was exposed. The upper half was distinctly divided from the lower half and appeared grossly normal, and a normal ureter appeared from this pole. The lower half of the kidney was hydronephrotic and the ureter was dilated. Both ureters were followed down for a considerable distance and freed. They were both contained in the same sheath. The lowermost ends of the ureters were exposed through a separate incision in the right lower quadrant of the abdomen. The dilated lower ureter was ligated and severed about 1½ cm. from its entrance into the bladder. The proximal ureteral stump was covered with a finger cot and then drawn through the track and out

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Fig. 3.—Case 1 (L. A.): (A) Retrograde pyelogram showing pyonephrosis of lower pole of double kidney with complete duplication of ureter. (B) Double retrograde pyelogram, showing rudimentary upper pelvis and calices and pyonephrotic lower pelvis. (C) Excretory urogram, showing good function of the remaining upper pole, three and one-half years postoperatively.



through the lumbar incision. The two halves of the kidney were then separated and the lower pole with its ureter removed.

Following operation there was some urinary leakage from the lumbar incision. Leakage also occurred from the anterior wound. This ceased after introducing an indwelling ureteral catheter; the patient was discharged with both wounds firmly healed.

Follow-Up.—December 28, 1935 (three years later): There was good function of the remaining upper pole of the right kidney, as evidenced by excretory urography (Fig. 3C).

Case 2.—A. T., male, age 40. *Diagnosis:* (1) Double right kidney with partial duplication right ureter. (2) Dendritic calculus lower pole. (3) Right hydronephrosis (both halves). *Operation:* (1) Right pyelolithotomy and nephrostomy (lower pole). (2) Right heminephrectomy (lower pole). *Result:* Well.

This patient entered the hospital August 29, 1933, complaining of right loin pain, frequency and pyuria of eight months' duration. There had been no chills or fever. His past history disclosed two cystolithotomies performed 21 and 15 years previously, and an incision and drainage operation for a left perinephritic abscess 13 years previously. Examination disclosed a pale individual. The right kidney was palpable and tender. The urine contained numerous white blood cells. Blood urea was 75.0 mg. per 100 cc., hemoglobin 55 per cent. Roentgenologic examination showed a dendritic calculus on the right side and a hydronephrotic, very much deformed pelvis on the left. Roentgenograms taken with the catheter in situ on the right side disclosed the tip of the catheter to be in contact with the calculus at the 25 cm. level. There was no flow of urine from this side. On the left side the excretion and function were poor. The azotemia was treated vigorously. Apparently the patient had a very much damaged kidney on the left side and a blockade on the right. It was felt his only chance lay in removing the calculus on the right side. Accordingly, despite the poor condition of the patient, the dendritic calculus was removed piecemeal through pyelotomy and nephrotomy incisions. Control roentgenograms taken on the operating table revealed no further calculi. A drainage tube was placed in the pelvis through a nephrostomy opening.

Postoperatively, the blood urea rose to 168.0 mg., and after intensive treatment the azotemia gradually cleared up. The patient drained moderately large amounts of urine through the nephrostomy tube. Blue was injected into this tube to determine the patency of the ureter, but none appeared in the bladder urine. This indicated some obstruction, and with this in mind, cystoscopy was performed. The right and left ureters were catheterized to the pelvis; good indigo carmine excretion was obtained from the right side (catheter apparently in the upper pole). Concentrated blue solution injected through this catheter did not appear through the nephrostomy tube. Blue coloration from the left side was definitely weaker than that obtained from the right, contradicting previous functional tests. A suspicion of a right double pelvis was entertained. This was confirmed when pyelograms were made singly and simultaneously and at different times, by injecting radiopaque solution through the right ureteral catheter and through the nephrostomy tube. About this time, the patient suddenly passed a small, almond sized stone. It was felt that this may have represented a portion of the original calculus which had broken off and had obstructed the flow from the nephrostomized lower pole. Indigo carmine solution was instilled through the nephrostomy tube, but did not appear in the bladder. The nephrostomy tube was inadvertently removed, and it was necessary to reoperate upon the patient and to renephrostomize the kidney. At this operation, it was impossible to thoroughly explore the entire region, and to demonstrate a double ureter. Heminephrectomy was considered at this operation, but it was felt the patient probably could not undergo such a formidable procedure and that we would have to be satisfied with a permanent nephrostomy. His blood urea remained at about 35.0 mg. Apparently the strongest functional element in his excretory system was the upper pole of the right kidney.

Follow-Up.—He was discharged with the nephrostomy tube in place. Three months

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later he returned to the hospital because of some trouble he was having in caring for the nephrostomy tube and its drainage. Studies showed practically no function from the lower half of his right kidney. In view of his better general condition and practically normal blood urea, it was decided to perform a heminephrectomy of the lower half of the right kidney.

Operation.—March 27, 1934: Heminephrectomy (A. Hyman). The right kidney was found densely adherent as a result of the two previous operations. The kidney was, however, eventually mobilized. It was impossible to expose the ureter. Somewhat more than the lower half of the kidney was resected and hemostasis effected by chromic sutures underpinned with fat. The patient was discharged 26 days after operation.

Follow-Up.—After discharge the patient developed urinary leakage from the wound, which has persisted, despite all local measures. It was felt the leak was caused by retained cortical renal tissue. Cystoscopy soon after discharge from the hospital (August, 1934) showed good indigo carmine excretion from the right kidney. An excretory urogram, taken in August, 1936, showed insufficient visualization for diagnostic purposes on both sides.

Case 3.—D. A., female, age 40. *Diagnosis:* (1) Double right kidney and partial duplication of right ureter. (2) Pyonephrosis and hydro-ureter, lower half. *Operation:* Heminephrectomy, lower half of right kidney. *Result:* Well.

Ten years previously, this patient had had colicky pains in the right lumbar region. Nine years ago, a right ureterolithotomy was performed. Seven years later, there was a recurrence of right-sided pain, pyuria and frequency of urination. On cystoscopy a single right ureter orifice was seen, and there was a temporary obstruction to the passage of a ureteral catheter at 9 cm., where a retention of 80 cc. of pus was found. At the 29 cm. level there was a retention of 20 cc. of pus. Retrograde pyelography revealed a duplicated right kidney with a normal, small upper pelvis and markedly dilated lower pole.

Operation.—Heminephrectomy (right lower half) and partial ureterectomy (L. Edelman): The lower pole was cystic, the pelvis was dilated and the ureter was one inch in thickness. The upper pole ureter appeared normal. There were two distinct sets of vessels going to both halves of the kidney. The dilated ureter was fixed in the bony pelvis to a cystic structure, which was probably an intraligamentous cyst. The ureter was ligated one inch from the bladder, where its diameter measured one-quarter of an inch. The actual site of the bifurcation could not be determined, because of the intimate relationship with the cyst. The lower portion of the kidney was then resected. The bleeding was readily controlled by a few mattress sutures of chromic gut.

Recovery was uneventful. It has been impossible to obtain a follow-up on this patient.

Case 4.—L. G., male, age 44. *Diagnosis:* (1) Horseshoe kidney. (2) Complete duplication of left half of horseshoe kidney. (3) Hydronephrosis upper half left kidney. (4) Pyonephrosis lower half left kidney. *Operation:* Heminephrectomy, lower half left side of horseshoe kidney. *Result:* Well.

The patient was admitted with a three weeks' history of left renal colic, associated with chills, fever, hematuria and pyuria. On examination, a large, smooth, tender mass was felt on the left side of the abdomen, extending from the free border of the ribs to the crest of the ilium. At cystoscopy the right ureter orifice was found to be normal. On the left side two ureteral orifices were present. All three ureters were catheterized without obstruction. There was thick, purulent urine coming from the left lower kidney, the function of which was very poor.

Operation.—September 10, 1912: Heminephrectomy lower half left side of a horseshoe kidney (L. Buerger). A large, multilocular, pyonephrotic sac, adherent to the peritoneum and involving the whole lower half of the left kidney, was found. The upper half of the left kidney represented the left half of a horseshoe kidney (Fig. 4). The sac contained two quarts of purulent material. The upper pole presented a hydronephrotic pelvis with good renal parenchyma. There were two ureters. The pyonephrotic half

of the left kidney was excised. At first the upper ureter was ligated and cut, with the intention of removing the entire left kidney, but due to the excellent condition of the parenchyma in the upper pole, it was decided to attempt to conserve this pole. Accordingly the cut upper ureter ends were anastomosed over a No. 6 Fr. ureteral catheter. The patient had some urinary leakage through the lumbar sinus.

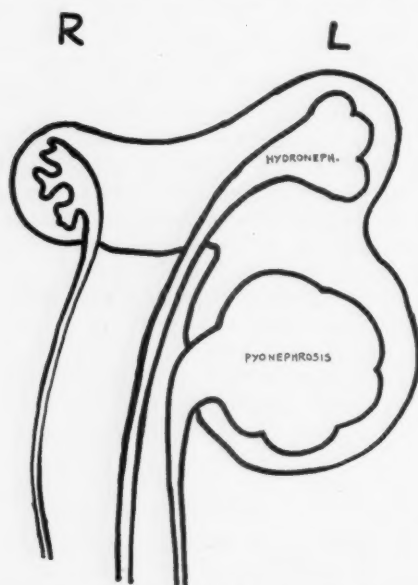


FIG. 4.—Case 4 (L. G.): Schematic drawing of operative findings in a case of horseshoe kidney with complete duplication of its left half, which joins the right kidney by the upper pole.

nephrectomy for calculous hydronephrosis, left upper pole (E. Beer). The kidney was found to be about one-third larger than normal and surrounded by adherent fat. Two ureters were exposed and found to unite three inches below the lower pole of the kidney. The lower ureter was normal; the upper ureter contained a stone at the level of the lower pelvis. It was thickened, as was the pelvis. The vessels were exposed, and it was noted that the branches to the upper third of the kidney apparently came off the main vessels. These upper branches were ligated. The parenchyma of the upper pole was reduced to about one third the normal amount and the pelvis was hydronephrotic. After ligating the vessel branches, the upper third of the kidney was cut across about 1 cm. above the groove which separated the healthy part from the hydronephrotic third. Bleeding was controlled with hemostatic sutures and the beveled edges of the kidney incision were brought together with chromic sutures, underpinned with fat. The upper ureter was tied 1 cm. above the bifurcation and removed in one piece, with the hydronephrotic pole. Except for a mild wound infection, the patient made a good recovery.

Follow-Up.—An excretory urogram, May 21, 1938, seven years after operation, showed good function of the remaining lower half of the left kidney (Fig. 5C).

Case 6.—L. F., female, age 42. *Diagnosis:* (1) Complete duplication left kidney and ureter. (2) Nephrolithiasis, lower half. *Operation:* Left heminephrectomy, lower half. *Result:* Died.

This patient had had recurrent attacks, over a period of eight years, of right lumbar pain associated with frequency, urgency and pyuria. She thought she had passed a small stone six months previously. Three months later she began to complain of left lum-

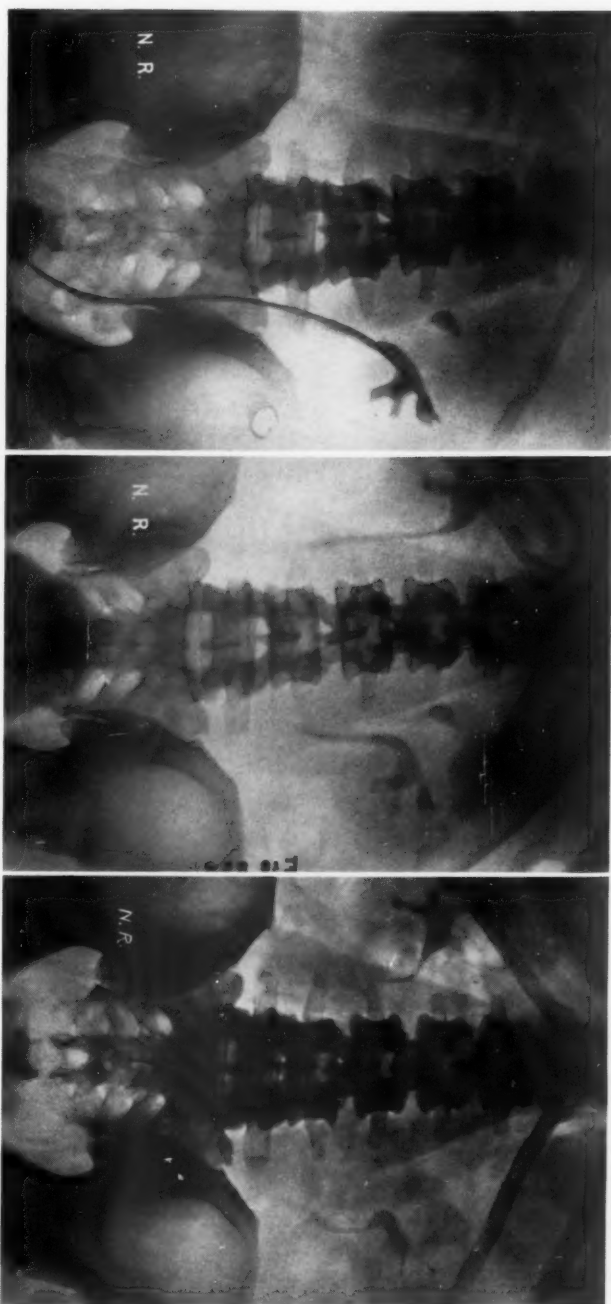
Follow-Up.—At cystoscopy, performed one month later, only a few drops of urine were obtained from the upper pole. There was also a trace of indigo carmine from the lumbar wound after 40 minutes.

The patient was still alive, about 17 years later, although no functional studies of the remaining upper pole have been made.

Case 5.—N. R., male, age 44. *Diagnosis:* (1) Double kidney (left) with partial duplication of the ureter. (2) Calculus, hydronephrosis left upper pelvis. *Operation:* Heminephrectomy, upper pole left kidney. *Result:* Well.

In 1906, patient had a cystolithotomy performed. For five years previous to admission to the hospital, he complained of bilateral sacral pain with recent radiation along the left thigh. There were no urinary symptoms. Cystoscopic and pyelographic studies revealed a double left kidney and partial duplication of the ureter, with calculous hydronephrosis of the upper pelvis (Fig. 5A and B).

Operation.—August 18, 1931: Hemi-



(A)

(B)

(C)

Fig. 5.—Case 5 (N. R.): (A) Retrograde pyelogram showing double left kidney with partial duplication of the ureter (one orifice in bladder). The catheter has found its way into the lower pole, which appears normal. The upper pole shows a calculus in the region of the ureteropelvic junction (C). (B) Excretory urogram showing a double kidney on the left with a stone at the ureteropelvic junction of the upper pelvis and a hydronephrosis of the upper segment (A). (C) Excretory urogram, seven years postoperatively, showing good function of the conserved lower two-thirds of the left kidney, with pelvis, calices and ureter well filled.

bar pain and hematuria. Roentgenologic examination demonstrated a large concretion about 1 cm. in size in the region of the lower pole of the left kidney. Cystoscopic and pyelographic studies showed a completely duplicated left kidney and ureter with a calculus in the lower pelvis.

Operation.—August 26, 1926: Heminephrectomy, left lower half for calculosis (P. W. Aschner). The kidney was partially divided into upper and lower half. A large stone and considerable sandy deposit were found in the lower half of the kidney. There were two sets of vessels, each going to the respective poles of the kidney. The lower half of the kidney was opened through a nephrotomy incision. The stones and sand material were removed, and the pelvis irrigated. In view of the presence of considerable sand material and the unlikelihood of successfully removing all of it, an heminephrectomy was

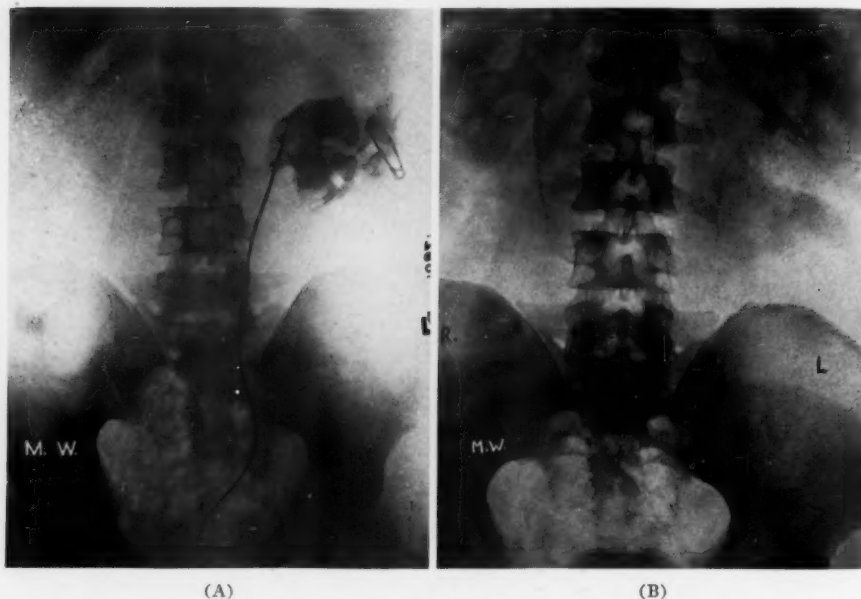


FIG. 6.—Case 7 (M. W.): (A) Complete duplication left kidney and ureter. Pyelogram showing pyonephrosis of lower half. The second ureter not catheterized, because of good function after injection of indigo carmine. (B) Excretory urogram, six years postoperatively, showing good excretion from remaining upper segment, pelvis of which is small and leads into a normal appearing ureter.

performed, during which procedure an aberrant vessel to the upper pole was injured and bled profusely. The hemorrhage was finally controlled by a hemostat, which was left in place, and the lower part of the kidney was resected. The patient lost a considerable amount of blood; she was returned to bed in shock, from which she died three hours later, despite all measures to combat this condition.

Case 7.—M. W., male, age 26. *Diagnosis:* (1) Complete duplication left kidney and ureter. (2) Pyonephrosis lower half (aberrant vessel). *Operation:* (1) Left nephrostomy, lower half. (2) Secondary heminephrectomy, left lower half. *Result:* Well.

For three weeks, the patient had complained of left lumbar pain and dysuria. The urine, which was said to have been clear at first, later became turbid, and a temperature of 103.8° F. developed. On physical examination there was found a ballotable, tender mass in the left lumbar region. There was definite costovertebral tenderness on this side, and roentgenologic examination revealed a curvature of the spine, with the convexity toward the right. The urine was loaded with pus. Excretory pyelography showed a normal kidney on the right side. The left pelvis and calices were deformed and the left ureter appeared compressed in its upper half. At cystoscopy two ureter orifices

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were found on the left side. The lateral one (lower pole) was slightly edematous. Milky fluid was seen discharging from this orifice. The lateral ureter was catheterized for 20 cm., where an impassable obstruction was encountered. There was no flow at this level. The mesial ureter orifice (upper pole) appeared normal, good indigo carmine efflux was seen, and it was not catheterized. A diagnosis of pyonephrosis of the lower half of a double kidney was made (Fig. 6A).

Operation.—December 12, 1931: Nephrostomy of a pyonephrotic lower half of a left double kidney (E. Beer). There was extensive perinephritis surrounding the lower pole. The lower pole, comprising about two-thirds of the kidney, was composed of three calices and had been converted into a sac. The ureter to the lower half was the size of a pencil. No calculus was present. The ureter could not be probed from above.

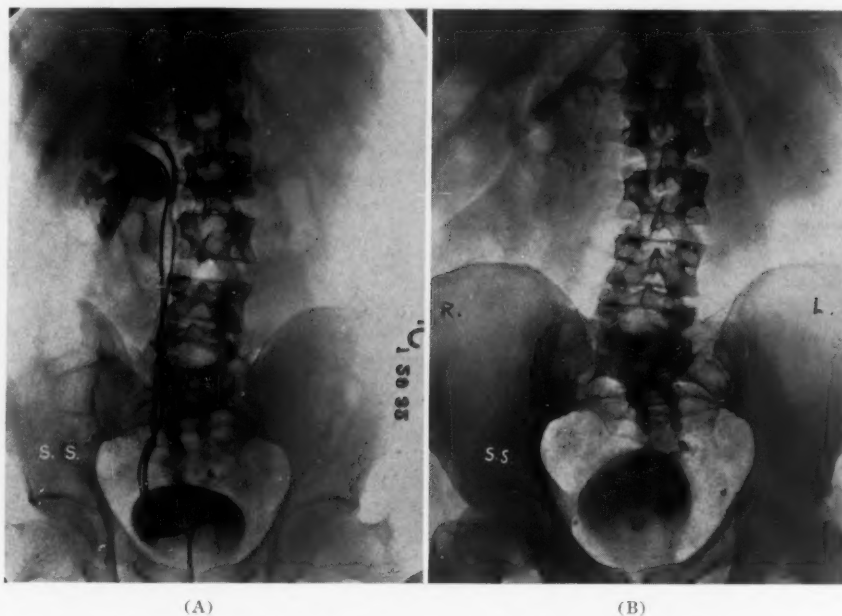


FIG. 7.—Case 8 (S. S.): (A) Retrograde pyelogram showing complete duplication with rudimentary upper pelvis and calculous pyonephrosis of lower pole, right kidney. (B) Excretory urogram, six years postoperatively, showing good function of remaining upper pole, right kidney.

There were about eight ounces of pus in the pyonephrotic sac. The exact cause of obstruction to the ureteral catheter at cystoscopy could not be determined.

Postoperatively there was very little discharge through the nephrostomy wound. Blue stained fluid instilled into the lower pelvis through the nephrostomy tube did not appear in the voided urine, indicating some form of blockade. Catheterization of this ureter and instillation of blue stained fluid did not show any flow out of the nephrostomy tube, but after digitally opening into the lower pole, there was copious flow of blue fluid. Iodide was instilled into the lower pole and a large dilated pelvis was outlined. No iodide passed down into the ureter. There was a defect at the ureteropelvic junction, suggesting an aberrant vessel. Excretory urography showed no excretion from this pole.

Second Operation.—January 2, 1932: Subcapsular heminephrectomy for pyonephrotic sac of lower pole (E. Beer). On delivering the sac, two groups of vessels were exposed going to the lower pole, and a branch apparently crossing the ureteropelvic junction and occluding same was found. The sac was the size of a peach, and the amount of renal parenchyma reduced. The ureter was small. The patient was discharged from the hospital well.

Follow-Up.—In January, 1938, six years after operation, excretory urography showed good function of the remaining upper pole (Fig. 6B).

Case 8.—S. S., male, age 47. *Diagnosis:* (1) Complete duplication right kidney and ureter. (2) Renal calculus and pyonephrosis lower half. *Operation:* Heminephrectomy, lower half right kidney. *Result:* Well.

Fifteen years previously this patient had had a suprapubic cystolithotomy performed. Eight years later, he developed a right renal colic and since then has had repeated attacks. During the year before his present admission, the pain has been persistent. He noted hematuria on one occasion. Cystoscopic and pyelographic studies showed a single normal kidney on the left side. The right kidney and ureter were completely duplicated. The lower half was filled with stones and was moderately dilated. The upper half was also dilated (Fig. 7A).

Operation.—February 5, 1932: Heminephrectomy, lower pole right kidney for calculus pyonephrosis (E. Beer). There were considerable perinephric and peri-ureteral adhesions. The lower ureter was moderately thickened. A large artery crossed posteriorly to the lower ureteropelvic junction, toward the lower pole. After evacuating many stones and much sand from the lower pelvis and calices, only a shell of kidney parenchyma was found in the lower pole. The upper half of the kidney had good parenchyma and the upper pelvis was found to be small. The lower ureter was isolated and ligated. After sectioning it, and the aberrant vessel at the ureteropelvic junction, the lower pole was resected. Three mattress sutures were used to control the slight ooze and to close the lower resected edge. Except for a wound infection, the patient made an uneventful recovery and was discharged well.

Follow-Up.—In April, 1938, six years later, excretory urography showed good function of the remaining upper pole (Fig. 7B).

Case 9.—M. O., male, age 23. *Diagnosis:* (1) Bilateral complete duplication of kidneys and ureters. (2) Hydronephrosis right upper pelvis. (3) Atrophy right kidney. (4) Left hydronephrosis and hydro-ureter, lower pole. *Operation:* (1) Right complete ureteronephrectomy. (2) Heminephrectomy, lower pole left kidney. *Result:* Well.

The patient was first seen in 1922, with a history that for a period of two years he had been complaining of bilateral lumbar pain, frequency, urgency, and burning on urination. A complete nephrectomy of a double right kidney and ureter was performed at that time. The entire kidney was found atrophic. The upper pelvis was dilated, due to a valve-like obstruction in the upper ureter. In view of the atrophy of the entire organ, complete nephrectomy and ureterectomy was performed, June 22, 1922, by Dr. A. Hyman.

The patient was again admitted to the hospital, in 1927, complaining of persistent pyuria since his previous discharge from the hospital. There was also dull, left lumbar pain. Cystoscopy showed four ureter orifices. The left lateral ureter (lower pole) was edematous and obstructed at 1 cm. The left mesial orifice (upper pole) was catheterized to the pelvis. A retrograde pyelogram of the upper pole showed a triangular shaped area of upper pelvis with two or three calices. No pyelogram could be obtained of the lower pole.

Operation.—October 14, 1927: Heminephrectomy and partial ureterectomy, lower half left kidney (A. Hyman). The upper half of the kidney appeared normal. The lower half was a hydronephrotic sac. There was a definite line of fusion across the middle of the kidney. The upper ureter was normal. The lower ureter was thickened and dilated to the size of a thumb. The lower pelvis was opened and a finger introduced into the calices, to demarcate the limits of the lower half. Because of bleeding, some mucosa was left behind. Mattress sutures, underpinned, were employed to control the bleeding.

Subsequent Course.—Postoperatively, the patient developed uremia and psychosis and an infected wound, from which he gradually recovered after intensive treatment. He has

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remained well since then for the past 11 years, with normal blood urea. At present he is living on *approximately one-sixth of the normal amount of kidney parenchyma*.

Case 10.—J. G., female, age 14. *Diagnosis:* (1) Bilateral, complete kidney and ureteral duplication. (2) Pyonephrosis of lower half left kidney. *Operation:* Heminephrectomy, lower half left kidney. *Result:* Well.

The patient had complained of attacks of "pyelitis," associated with pyuria and fever since birth. After a number of years of investigation by numerous urologists, her case was finally diagnosed as one of complete bilateral kidney and ureter duplication, with the focus of her pyuria located in a pyonephrotic lower half of the left kidney.

Operation.—September 29, 1937: Heminephrectomy of the pyonephrotic lower half of the left kidney (E. Beer). Lower half of left kidney found to be markedly adherent.



FIG. 8.



FIG. 9.

FIG. 8.—Case 10 (J. G.): Excretory urogram showing good function in the remaining upper segment of left kidney, seven months after heminephrectomy of lower half of left kidney for pyonephrosis.

FIG. 9.—Case 11 (R. W.): Excretory urogram showing good function from remaining upper segment left kidney, one and one-half years after removal of left lower pole for pyonephrosis.

The ureter to the lower half was dilated and thickened. Several aberrant vessels were found, and these apparently contributed to the causation of the pyonephrosis. The renal cortex in the involved pole had practically disappeared, so that this portion of the kidney was converted into a sac. The upper pole contained good renal parenchyma. Because of the extreme thinness of the pyonephrotic sac, it was inadvertently opened during the separation of it from the upper pole. The contents of the sac were evacuated and the sac wall removed by sharp dissection and curettage. The ureter was divided low.

Subsequent Course.—Postoperatively, the wound became grossly infected. It was widely opened and allowed to heal by secondary intention. No hernia of the wound has occurred thus far. Excretory pyelography, eight months later, showed good function in the remaining upper pole (Fig. 8).

Case 11.—R. W., female, age 8. *Diagnosis:* (1) Complete duplication of left kidney and ureter. (2) Pyonephrosis of lower half left kidney. *Operation:* Left uretero-heminephrectomy, lower half. *Result:* Well.

For two weeks prior to admission to the hospital, the child complained of left loin pain and dysuria associated with cloudy urine and temperature, reaching as high as

105° F. On physical examination, the outstanding physical sign was left costovertebral angle tenderness. At cystoscopy, two left ureteral orifices were found, and a diagnosis of pyonephrosis of the lower half of the left kidney was established.

Operation.—July 3, 1936: Left, complete ureteroheminephrectomy of the lower half (E. Beer). The operation was carried out through a left loin and an anterior abdominal incision. By employing the two incisions it was possible to accomplish a complete ureterectomy of the ureter going to the lower half of the left kidney. The uppermost calix from the lower pole ran up into the upper pole for almost an inch. This was dissected out without entering the upper pole pelvis. The upper three-eighths of the kidney was normal, with apparently a normal ureter. The lower five-eighths of the kidney had a thin, mushy cortex. There were two sets of vessels. The ureter to the lower pole was dilated to within an inch of the bladder. Lower down, the two ureters were found to be contained in the same sheath and were difficult to separate. It was believed that some injury was done to the normal ureter during the separation, as, following the operation, there was leakage from both wounds for a time. The lumbar wound ceased discharging in six days. A catheter was passed to the upper pole of the kidney. Instillation of colored hippuran into this pelvis refluxed through the ureterotomy wound. The catheter was left indwelling for a short time and then removed. The leakage ceased and the patient was discharged from the hospital well.

Follow-Up.—Four months later, an excretory urogram was made, which showed a well functioning residual upper pelvis (Fig. 9). Sixteen months later, at cystoscopy, good indigo carmine and clear urine were obtained from the remaining renal segment.

Case 12.—A. G., male, age 40. *Diagnosis:* (1) Complete duplication of left kidney and ureter. (2) Infected hydronephrosis, lower half left kidney. (3) Hydronephrotic, contracted upper left kidney. *Operation:* Heminephrectomy, left upper pole. *Result:* Well.

For 12 years previous to admission, this patient had had attacks of left loin pain, associated with pyuria. At cystoscopy, a large intruding prostate was found, which made catheterization of the ureters difficult. The right ureteral orifice was normal, and there was good kidney function on this side. On the left side, two ureteral orifices were found. One of these was catheterized and an obstruction was met at the 9 cm. level. This was readily overcome and the catheter passed up into the kidney. Twenty-five cubic centimeters of turbid urine, containing a fair amount of indigo carmine, were obtained. A pyelogram made through this catheter showed dilatation of the calices in the lower pole of a double kidney.

Operation.—January 15, 1935: Heminephrectomy, left upper pole (E. Beer). The upper pole of the left kidney was found to be collapsed and led into a separate thickened ureter. The lower two-thirds of the kidney parenchyma felt normal. The upper pole was resected. The cut parenchyma was sewn over with underpinned chromic sutures. The exact nature of the obstruction of this portion of kidney could not be established. It may have been caused by some abnormal vessel, which had not been identified during the operation. The patient was discharged from the hospital fully recovered.

Follow-Up.—Thirty-two months later the patient was still well and had no complaints. It has not been possible to get the patient to return recently for check-up on the function of the remaining lower pole.

Case 13.—C. W., female, age 15. *Diagnosis:* Complete duplication of left kidney and ureter with ectopic ureter opening just below urethral meatus. *Operation:* Heminephrectomy, upper pole left kidney; partial ureterectomy. *Result:* Well.

The patient gave a history of constant dribbling of urine since childhood. No other urinary symptoms, except frequency. The rest of the past history was irrelevant. Abdominal examination was negative. Just below the urethral meatus in the midline was found a small, teat-like prominence with a tiny opening in it. A small ureteral catheter could be passed into this orifice for only 2 cm. The urine which dripped from the opening was clear. The vagina and vulva were reddened. Rectal examination was

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negative. Roentgenologic examination showed no spina bifida. At cystoscopy, the bladder and both ureteral orifices were found to be normal. Both ureters were catheterized without obstruction. Clear urine was obtained from both kidneys. The bladder was filled with blue stained solution and a cotton pad placed in the vagina, which became wet, but was not stained blue. This finding ruled out the possibility of a vesicovaginal fistula. The opening below the urethral meatus was then injected with 20 cc. of sodium iodide. This was found on roentgenologic examination to have filled the upper pole of a double left kidney. The lower pole was filled by means of a catheter in the left ureteral opening in the bladder (Figs. 10A and B).

Operation.—September 14, 1928: Left heminephrectomy, upper pole (A. Hyman). The left kidney was exposed and found to be about normal in size. There was a double pelvis, the upper one being markedly hydronephrotic. The upper pole of the kidney was

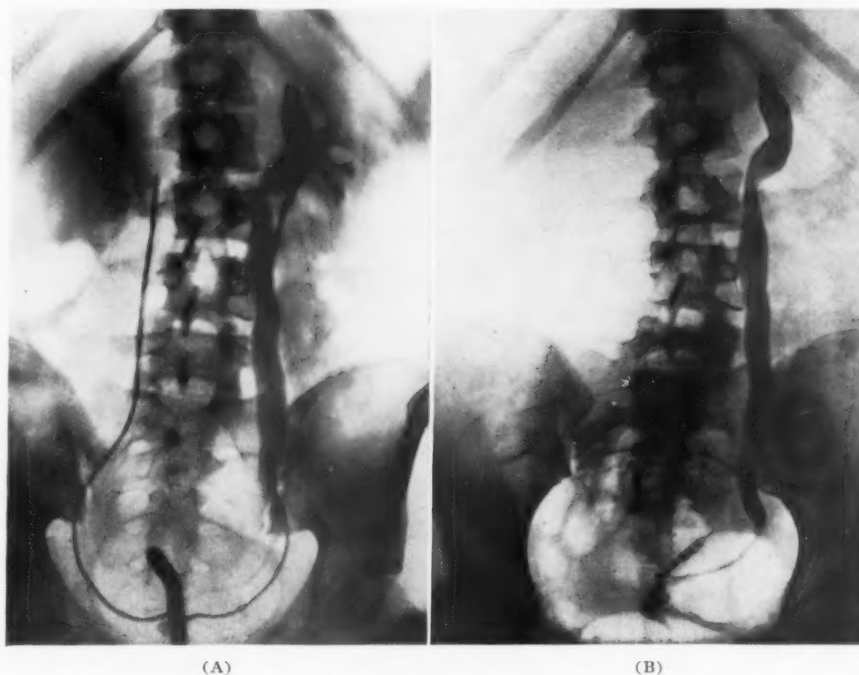


FIG. 10.—Case 13 (C. W.): (A) Retrograde ureteropyelogram showing an ectopic ureter on the left side, opening just below urethral meatus. Double pyelogram showing normal lower pole with ectopic ureter running toward upper pole. (B) Retrograde ureterogram, filling only the ectopic ureter, showing marked dilatation (ref. A).

resected, as well as the ureter entering the upper pelvis, down to within three inches of the bladder. The postoperative course was uneventful.

Follow-Up.—When seen two months later, the patient had no further dribbling. Cystoscopy revealed strong indigo carmine efflux from both ureteral orifices. Eight months later, the patient had no symptoms.

Case 14.—S. S., female, age 32. *Diagnosis:* (1) Right double kidney with ectopic ureter. (2) Pyonephrosis, left upper pole. *Operation:* Heminephrectomy, left upper pole. *Result:* Well.

The patient was admitted to the Gynecologic Service two months previously, because of urinary incontinence and frequency, which had followed childbirth eight years previously. She had had marked frequency of urination during cold weather since childhood. Anterior and posterior colporrhaphy and parametrial fixation were performed. On the

fifth postoperative day her temperature rose to 103.6° F. On the eighth day, she began to complain of right loin pain. The urine contained a great deal of pus. A tender mass was palpable in the region of the right kidney. This mass rapidly increased in size. Excretory urography revealed a normal, low lying pelvis of the right kidney (Fig. 11A) with a large mass to the right of the spine, obscuring the psoas margin. A diagnosis of a retroperitoneal abscess was made.

First Operation.—November 12, 1937: Incision and drainage (Dr. A. Hyman). A large encapsulated mass was found. It was opened, and a great deal of thick pus was evacuated. The nature of the mass could not be determined by inspection alone, and a section of the membrane wall was taken for microscopic examination. The pathologic report was "fragment of atrophic kidney tissue with pelvic mucosa, probably a portion of an hydronephrotic sac."

Postoperative Course.—Following this procedure the patient's temperature dropped to normal and remained so. The discharge from the wound became minimal, and there remained only a small fistulous tract, which was kept open for further investigation. Indigo carmine, administered intravenously, was not discharged through the fistulous opening. Blue stained hippuran was instilled through the tract, causing the patient to have a desire to urinate after two ounces had been instilled. She was then catheterized and an ounce of unstained, clear urine was obtained. Obviously, therefore, there was no connection between the cavity above and the bladder. Cystoscopy was performed. A single right ureter was catheterized for a few inches with a Garceau catheter (Fig. 11B). With hippuran instilled into the fistulous tract at the same time as the Garceau catheter was injected, a simultaneous roentgenogram was obtained (Fig. 11C) which showed that we were dealing with a double kidney and double ureter, and that the drainage operation previously performed had drained a pyonephrosis of the upper pole. Both pelves were dilated. The ureters were tortuous and swung to the right in their mid-portions. The ultimate termination of the ureter going to the upper pole could not be identified (only a single right and left ureteral orifice were seen at cystoscopy). The patient was discharged with the fistulous opening. She returned one month later, having gained ten pounds during the interim, and with the history that she was having attacks of right loin pain. Cystoscopic check-up again revealed only a single right and left ureteral opening. Pyelographic studies corroborated the findings obtained previously. Inspection of the external genitalia and genito-urinary tract did not reveal an ectopic ureteral opening after instillation of blue-stained solution into the upper pole of the kidney through the fistulous tract. However, pressure over the right vaginal wall caused a reflux spurting of the blue solution through the fistula. It was then fairly obvious that we were dealing with an instance of double kidney with an ectopic, right ureteral implantation, although the ureter opening was not seen.

Second Operation.—January 4, 1938: Heminephrectomy, right upper pole, and partial ureterectomy down to the iliac vessels (E. Beer). The kidney was exposed. The ureters were adherent to the anterior portion of the previous wound. One ureter was 2 cm. in diameter and very much thickened. The other was slightly larger than normal, soft and healthy looking. After identifying the two ureters, they were followed up toward the kidney and the normal appearing one was found to enter the lower two-thirds of the kidney, whereas, the thick ureter entered the upper pole. The upper pole was separated from the lower pole, using one finger in the upper pyonephrotic sac as guide for the line of separation. The vascular supply to the lower part of the kidney was left intact; very little vascular pedicle was identified, going to the atrophic and pyonephrotic upper pole. Following the separation of the upper pole, the bleeding from the cut kidney surface was controlled by three or four underpinned chromic sutures. The large ureter was then dissected free, down to the iliac vessels, and carefully separated from the other ureter, which was contained in the same sheath. The diseased ureter was tied at the level of the iliac vessels and divided with a carbolized knife. It was not

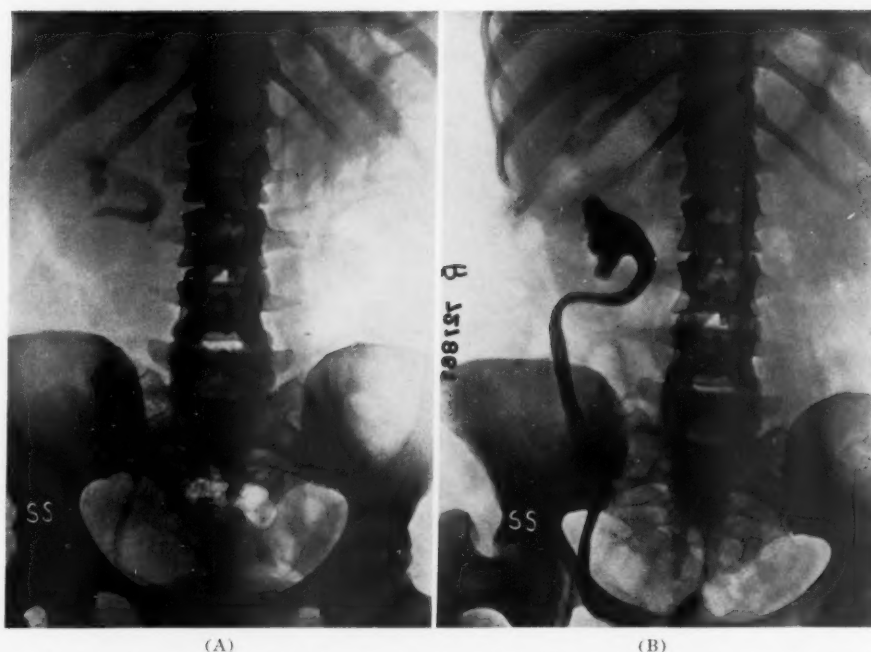


FIG. 11.—Case 14 (S. S.): (A) Excretory urogram showing lower pole of double kidney, no excretory media from upper pole (ref. B and C). (B) Retrograde pyelogram showing outline of lower pole of duplicated right kidney (ref. A and C).

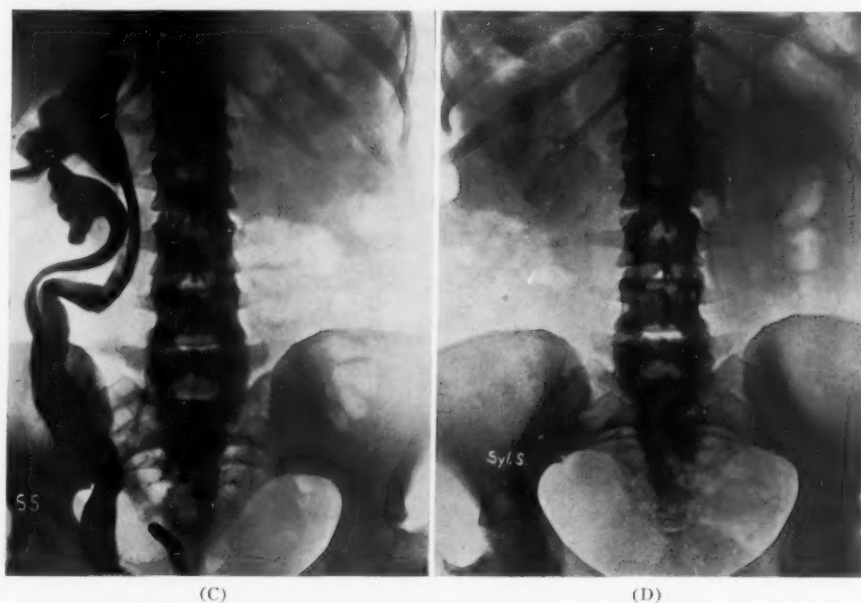


FIG. 11.—(C) Roentgenogram taken after simultaneous instillation of radiopaque solution through the nephrostomy tube and through the ureteral catheter, showing double kidney (right) and ureters. Note the dilatation of the upper pelvis and its catheter, which was found to end blindly in the right anterior vaginal wall. (D) Excretory urogram four months postoperatively, showing good function of the conserved upper two-thirds of the right kidney. Right ureter can be seen as in the original pictures, displaced far from the spine.

TABLE VI

SYNOPSIS OF 14 CASES OF HEMINEPHRECTOMY, OPERATED UPON FOR DISEASE OF A DOUBLE KIDNEY

Case No.	Sex	Age	Anomaly	Pathology	Operation	Pole Involved	Follow-Up on Remaining Pole
1. L. A.	F.	29	Right double pelvis and ureter, complete duplication	Pyonephrosis, right lower half	Right heminephrectomy; complete ureterectomy	Lower	3½ years, good function
2. A. T.	M.	40	Right double pelvis, partial duplication of ureter	Dendritic stone, right lower pole. Hydronephrosis, both right pelvises. Left hydronephrosis	Right pyelolithotomy, lower pole nephrostomy; heminephrectomy, lower pole	Lower	Leakage from wound
3. D. A.	F.	40	Right double pelvis, partial duplication of ureter	Pyonephrosis, right lower pole	Right heminephrectomy; ureterectomy, to 1 inch from bladder	Lower	No follow-up
4. L. G.	M.	44	Horseshoe kidney, complete duplication left kidney and ureter	Hydronephrosis, left upper pole. Pyonephrosis, left lower pole	Heminephrectomy, lower half left horseshoe kidney. Ureteral anastomosis (ureter going to upper pole inadvertently injured)	Lower	Some urine from upper pole 1 month later, no further follow-up
5. N. R.	M.	44	Left double pelvis, partial duplication of ureter	Calculus hydronephrosis, left upper pole	Heminephrectomy, left upper pole	Upper	7 years, excretory urogram, good function
6. L. F.	F.	42	Complete duplication left kidney and ureter	Nephrolithiasis, left lower pole	Heminephrectomy, left lower pole	Lower	Died 3 hours postoperatively; shock
7. M. W.	M.	30	Complete duplication left kidney and ureter	Pyonephrosis, left lower pole (aberrant vessel); perinephritis	Heminephrectomy, left lower pole	Lower	7 years, excretory urogram, good function

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8. S. S.	M.	47	Complete duplication right kidney and ureter	Calculus, lower pole right kidney	Heminephrectomy, right lower pole	Lower	5 years, excretory urogram, good function
9. M. O.	M.	23	Bilateral, complete duplication kidneys and ureters	Hydronephrosis, right upper pole. Atrophy, right kidney. Hydronephrosis, left lower pole with hydro-ureter	Right nephro-ureterectomy (complete). Heminephrectomy, left lower pole	Lower	11 years postoperatively, patient still living, good function
10. J. G.	F.	10	Bilateral, complete duplication kidneys and ureters	Pyonephrosis, left lower pole	Heminephrectomy, left lower pole	Lower	8 months postoperatively, good function
11. R. W.	F.	8	Complete duplication left kidney and ureter	Pyonephrosis, left lower pole	Heminephrectomy, left lower pole; complete ureterectomy	Lower	1 1/2 years postoperatively, good function
12. A. G.	M.	40	Complete duplication left kidney and ureter	Infected hydronephrosis, left lower pole. Hydronephrotic contracted, left upper pole	Heminephrectomy, left upper pole	Upper	2 years postoperatively, no symptoms
13. C. W.	F.	15	Complete duplication left kidney and ureter. Ectopic left ureter (urethra)	Ectopic ureter going to upper pole	Heminephrectomy, left upper pole	Upper	2 months postoperatively, strong indigo carmine both kidneys. 3 years later no complaints
14. S. S.	F.	32	Right double kidney (ureter to upper pole apparently ending blindly in anterior vaginal wall)	Pyonephrosis, right upper pole	Heminephrectomy, right upper pole	Upper	4 months postoperatively, excretory urogram, good function

considered advisable to remove the entire ureter, since this procedure would have entailed making a second incision in the abdominal wall, with certain resultant contamination from the loin wound. It was appreciated that fluid might collect in the remaining portion of the ectopic ureter and that it would probably present itself as a cystic dilatation in the right anterior vaginal wall, which could be dealt with subsequently by a simple incision.

Subsequent Course.—This cystic dilatation, corresponding with the lower stump of ureter, did occur in the vaginal wall, associated with fever. Incision and drainage was performed and pus was evacuated. The cavity was kept open for a number of days, and the patient discharged for further dressings in the dispensary. Four months postoperatively, an excretory urogram showed good function of the remaining lower pole (Fig. 11D).

Recapitulation of the Present Series of 14 Cases.—In our experience with 14 cases of heminephrectomy, there was a single operative mortality (Case 6), or 7 per cent. The disease was limited to the upper pole in four cases (28 per cent), and of these four cases, two had ectopic ureters. The upper pole and its ureter were removed, preserving for the patient the lower half of the kidney. In ten cases, the lower pole showed involvement, and in these the lower half was removed, allowing the upper portion to remain. The nature of the lesion in six cases was pyonephrosis; in five cases it was hydronephrosis; one case had a calculous hydronephrosis; in two cases there were multiple calculi. In none of our cases was it necessary to perform a secondary nephrectomy of the residual portion.

Stone¹² calculated that in a series of 30 collected cases, 10 per cent required secondary nephrectomy. It is also of interest to note that in 12 of the 42 nephrectomies reviewed by Eisendrath,³¹ there was no abnormality in one segment; in other words, heminephrectomy would have been the more conservative procedure. In five additional cases in his series, technical difficulties prevented a planned heminephrectomy, and a complete nephrectomy was performed.

In those cases which we have been able to follow up by cystoscopy or pyelography, all of them showed good function of the remaining portion of kidney at variable lengths of time after the operation.

It is evident, therefore, from a study of these cases that conservatism is most important, and that kidney tissue should be saved whenever possible. This point may be emphasized by reference to Case 9, who has been living for 11 years on what amounts to approximately one-sixth of the normal amount of kidney parenchyma.

SUMMARY AND CONCLUSIONS

(1) A series of 104 cases of double kidney is presented and an analysis of these cases emphasizes the well known statement that an anomalous kidney is prone to disease.

(2) Sixty per cent of these cases had symptoms, and 25 per cent required some operative procedure.

(3) Fourteen of these cases required a heminephrectomy, which is discussed from the standpoint of indications, contraindications and technic.

(4) The conclusion is reached, after follow-up study of these 14 cases,

that conservation of renal tissue is indicated in instances of double kidney, and that heminephrectomy fulfils this purpose. Of the 14 cases of heminephrectomy, ten were adequately controlled by cystoscopy, excretory urography or both; the residual part of the kidney was found to function satisfactorily.

BIBLIOGRAPHY

- ¹ Braasch, W. F., and Scholl, A. J.: Pathological Complications with Duplication of the Renal Pelvis and Ureter (Double Kidney). *Surg., Gynec., and Obstet.*, **35**, 401, 1922.
- ² Braasch, W. F.: The Clinical Diagnosis of Congenital Anomaly in the Kidney and Ureter. *ANNALS OF SURGERY*, **56**, 726, 1912.
- ³ Israel: *Berlin Klin. Wchnschr.*, **45**, 1918.
- ⁴ Joseph, quoted by Burghel: *Lyon Chir.*, **30**, 434.
- ⁵ Mann, L. T.: Personal communication.
- ⁶ MacKenzie, D.: Personal communication.
- ⁷ Gruber: *Handbuch der Urol.*, iii, 1925. *Ztschr. f. Urol. Chir.*, **26**, 1919. *Ztschr. f. Urol. Chir.*, **7**, 1921.
- ⁸ Moulonquet: *Bull. et Mém. de la Soc. Nat. de Chir.*, **59**,² 956, 1933. Discussion of Papin's¹⁰ paper.
- ⁹ Ionel, T.: Sur l'anatomie et le Diagnostic du Rein Double. *Jour. d'Urologie*, **40**, 38, 1935.
- ¹⁰ Pearlman, S. J.: Heminephrectomy and Partial Ureterectomy for Fused Kidney and Double Ureter. *Urol. and Cut. Review*, **38**, 309, 1934.
- ¹¹ Hess, E.: Heminephrectomy. *Jour. Urol.*, **38**, 43, 1937.
- ¹² Stone, E.: Heminephrectomy. *Jour. Urol.*, **28**, 301, 1932.
- ¹³ Kretschmer, H. L.: Resection of Kidney. *Surg., Gynec. and Obstet.*, **60**, 984, 1935.
- ¹⁴ Goldstein, A. E., and Shaw, C. C.: Bilateral Supernumerary Fused Kidneys with Bilateral Reduplication of Ureters. *Urol. and Cut. Review*, **41**, 459, 1937.
- ¹⁵ Scher: A Case of Heminephrectomy for Hematuria. *Brit. Jour. Urol.*, **7**, 264, 1935.
- ¹⁶ Papin, E.: Incontinence d'urine par Uretère dédoublé Ectopique: Heminephrectomie. *Bull. et Mém. de la Soc. Nat. de Chir.*, **59**,² 954, 1933.
- ¹⁷ Vredensky: Resection of Lower Segment of Kidney for Hydronephrosis in a Child with Hereditary Double Kidney. *Sovet. Kher.*, **2**, 130, 1935.
- ¹⁸ Hicks, J. B.: Resection of Supernumerary Fused Kidney. *Surg. Clin. North Amer.*, **12**, 745, 1932.
- ¹⁹ Bagar, B.: Un Cas d'hemi-nephrectomie pour Rein Double avec Hydronephrose de la Moitié Inférieure. *Acta. Chir. Scandinav.*, **71**, 75, 1932.
- ²⁰ Brody, M. S.: Infected Supernumerary Ureter and Pelvis of Kidney: Heminephrectomy. *ANNALS OF SURGERY*, **98**, 119, 1933.
- ²¹ McClelland: Aberrant Ureters Causing Incontinence. *Brit. Jour. Urol.*, **4**, 46, 1932.
- ²² Campbell, M. F.: Ectopic Ureteral Orifice. *Surg., Gynec. and Obstet.*, **64**, 22, 1937.
- ²³ Legueu, F.: Necker Clinics, second series, 243, 1922. *Bull. de l'acad. de Méd.*, **84**, 213, 1920.
- ²⁴ Heymann: *Deutsche Med. Wchnschr.*, **38**, February 15, 1912. *Vereins beitrage*, 344.
- ²⁵ Beer, E.: The Use of Fat to Prevent Sutures Cutting into Parenchymatous Organs by Underpinning. *Surg. Gynec. and Obstet.*, **37**, 694, 1923.
- ²⁶ v. Lichtenberg, A.: Plastic Surgery of the Renal Pelvis and Ureter. *J.A.M.A.*, **93**, 1706, 1929.
- ²⁷ Lennander, K. G.: *Arch. f. klin. Chir.*, **12**, 471, 1900.
- ²⁸ Wright, B. W.: Heminephrectomy. *Urol. and Cut. Review*, **36**, 592, 1932.
- ²⁹ Lowsley, O.: *N. Y. State Jour. Med.*, **36**, 591, 1936.
- ³⁰ Miller, I. D.: Double Renal Pelvis with Single Hydronephrosis Treated by Ureter Ligation. *M. J. Australia*, **23**, 435, 1936.
- ³¹ Eisendrath, D.: Double Kidney. *ANNALS OF SURGERY*, **77**, 531, 1923.
- ³² Priestley, J. T., and Schulhof, M. G.: Heminephrectomy for Duplicate Kidney and Ectopic Ureter. *Proc. Staff Meet. Mayo Clinic*, **12**, 39, September, 1937.

DENERVATION OF THE BLADDER FOR RELIEF OF INTRACTABLE PAIN

W. J. MERLE SCOTT, M.D., AND CARLISLE F. SCHROEDER, M.D.

ROCHESTER, N. Y.

FROM THE DEPARTMENT OF SURGERY, THE UNIVERSITY OF ROCHESTER, SCHOOL OF MEDICINE AND DENTISTRY,
ROCHESTER, N. Y., AND THE DEPARTMENT OF UROLOGY, THE GODWIN JENNINGS HOSPITAL, DETROIT, MICH.

INTRACTABLE bladder pain associated with frequency and tenesmus is always a very distressing condition. When, however, it continues steadily for months at a time, it may completely undermine both the morale and the recuperative powers of the patient. There are two chronic diseases, namely, tuberculosis of the bladder and interstitial cystitis, in which this distressing syndrome is particularly apt to occur in a form which fails to respond to the usual methods of conservative treatment. Tuberculous cystitis is nearly always secondary to tuberculosis of the kidney. Consequently when the major infection is limited to one kidney, its removal generally allows the tuberculous involvement of the bladder to clear up. When both kidneys are involved in open lesions, however, there is a continuous and inevitable reinfection of the bladder, which frequently makes ineffective all measures directed toward the alleviation of symptoms from the local tuberculous ulceration. The effect of the pain and loss of sleep in patients combating a tuberculous involvement is distinctly harmful. In interstitial cystitis also, although many cases respond favorably to conservative measures, there are others in which the whole gamut of such procedures leaves the agonizing symptoms unaffected. Thus one of the cases which we saw had been treated energetically for a year by a very competent urologist who used all of the methods usually employed. Yet, in spite of this conscientious treatment, the patient was in constant pain; voiding every ten or 15 minutes, day and night, haggard from the loss of sleep, his morale completely shattered and having sustained a weight loss of 25 pounds. His actual survival depended upon obtaining relief.

Confronted with such examples of intractable vesical pain, it is quite comprehensible that numerous attempts have been made to produce an effective sensory denervation of the bladder. The great obstacle in the way of the successful accomplishment of this aim has been the threefold pathway of afferent impulses from the bladder: namely, the pelvic nerves (parasympathetic), the hypogastric nerves (sympathetic) and the internal pudendal nerves (somatic). Although all three of these routes probably do carry painful sensation from the bladder, and would all have to be interrupted in order to produce a complete sensory denervation of the bladder, they are not of equal clinical importance in the relief of intractable pain, nor, fortunately, is it necessary to interrupt them completely in order to alleviate the distressing symptoms.

Among the early attempts to control intractable bladder pain, the most drastic was the extirpation of the hypogastric ganglion. This procedure, of

course, produced a total paralysis of the emptying power of the bladder and consequently was quickly abandoned. The relief of other forms of pelvic pain, particularly those associated with the uterus, had been attempted with a considerable degree of success by the Lyonnais surgeons, Leriche and Cotte, and the technic of such sympathetic denervation by excision of the superior hypogastric ganglion has been fairly well standardized. The Italian surgeon, Pieri,¹ was the first to successfully carry out this procedure for the relief of bladder pain in two cases of tuberculous cystitis. Since that time, there have been scattered reports of individual cases or small groups of cases treated in this manner. Learmonth and Braasch² assembled 12 cases of vesical pain treated by sympathetic neurectomy. Only one of these cases was due to tuberculosis. Douglass³ reported five cases of interstitial cystitis treated in this manner. The most striking thing in regard to the results, in the few scattered cases recorded in the literature, is the marked variability in the degree of relief afforded the various patients. Thus one would obtain almost complete relief from excruciating pain while in another it would continue largely unabated. Learmonth⁴ stated that he had not yet been able to recognize the factors that made for success in some cases and failure in others.

Another idea suggested in the literature was that the improvement obtained in the favorable cases lasted only a few months and was not permanent. There were no data in the cases reported concerning any results later than a few months after operation. It was to gain further knowledge on these two important points that the present study was undertaken. Our series includes 11 patients with intractable bladder pain which we have sought to relieve by partial denervation of the bladder. Eight of these patients had tuberculous cystitis and three had interstitial cystitis.

The extreme variation that is found in the topographic anatomy of the sympathetic nervous system is generally appreciated. Therefore, the first point to be considered was that such a variation accounted for the lack of uniformity in the results obtained. Or, in other words, that the unsuccessful cases did not have complete interruption of the sympathetic pathways. When the operation is carried out by the standardized technic for the resection of the superior hypogastric plexus (Cotte's operation), there were two particular sources of error which we had learned from our previous experience in attempting to control other types of pelvic pain. In some cases fibers extend from the inferior mesenteric plexus to one of the separate hypogastric nerves, especially the left one, well below the bifurcation of the plexus. These are interrupted only by lifting up and freeing the posterior surface of the inferior mesenteric artery and its continuation as the superior hemorrhoidal artery deeply into the pelvis, and dividing all fibers extending backward and downward from it. Another set of fibers was found fairly frequently, which escaped division by the Cotte type of operation. These were sympathetic nerve fibers from the hypogastric ganglion or lower parts of the hypogastric nerves running upward and laterally directly to the sacral sympathetic chain on one side or the other which they then joined. Douglass

has also observed such branches going directly to the pelvic sympathetic chain and Pieri in his second paper advised ramisection of this chain presumably in order to interrupt such pathways.

As our first procedure, then, we extended the sympathetic interruption to include not only excision of the superior hypogastric plexus but also those fibers from the distal part of the inferior mesenteric and superior hemorrhoidal artery, and also those fibers going directly to the sacral chain. We accomplished the latter, not by attempting the hopeless task of dissecting out the individual fibers retroperitoneally very deep in the pelvis, but by removing the upper part of the sacral sympathetic chain including the first and second sacral ganglia. (The dissection must not be carried so deeply that



CHART 1.—Case 3. Showing, graphically, the result of sympathetic denervation in tuberculous cystitis.

the sacral [parasympathetic] nerves are injured.) In our second case, a young man, with bilateral renal tuberculosis and tuberculous cystitis, the left sacral chain was not disturbed for technical reasons. We found that this patient had much more pain than the succeeding ones on distention of the bladder after operation. Consequently, in subsequent cases we have been careful wherever possible to remove the upper sacral chain.

In the eight cases of tuberculous cystitis, sympathetic denervation has resulted in a marked decrease in the amount of pain suffered, often with an increase in bladder capacity and a decrease in the frequency of voiding. Case 3 illustrates the marked improvement obtained (Chart 1). This patient had been suffering so severely from pain and frequency that she had been

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on constant catheter drainage for eight months. Her bladder capacity was approximately 100 cc. Within four days after operation, her pain and frequency had markedly diminished. Her bladder capacity gradually increased, aided by dilatation, to 350 or 400 cc. and now three years after operation, she is having practically no bladder pain. The other cases of tuberculous cystitis have also shown a marked relief from pain. On the other hand, the cases of interstitial cystitis have shown a much more varied type of response. One patient showed a most striking relief of pain, one a moderate relief and one very little improvement.

We concluded, therefore, that in tuberculous cystitis, sympathetic denervation of the bladder could be relied upon to relieve the pain to a marked extent with a variable increase in bladder capacity, while in interstitial cystitis, the result was much more uncertain.

This difference in the results achieved by sympathetic denervation in tuberculous and interstitial cystitis could not be explained on the basis of individual variation in the nervous pathways. The only explanation that seemed reasonable for this discrepancy was in the site of the bladder involvement. In tuberculous cystitis, the chief area of irritation is in the trigonal region, whereas, in interstitial cystitis, it was more apt to be in the fundus of the bladder. Just how sympathetic denervation acts to relieve the bladder pain is uncertain, but as Learmonth⁵ and Cheetham⁶ reported, there is regularly a diminished tonicity in the trigonal region and a relaxation of the internal sphincter following sympathetic denervation. The mechanism of the production of visceral pain is not finally settled, but in the bladder as in other viscera, as Learmonth⁷ states, the majority of observers are agreed that pain arises as the result of tension in the viscus, whether from distention, from inability to evacuate its contents, or from incoordination of the muscular contraction. Consequently in the cases relieved of pain following sympathetic denervation, it is uncertain whether the relief is due to actual interruption of afferent pathways over which pain is carried or whether this result is due to the relaxation of the trigone and internal sphincter, the contraction or spasm of which would otherwise cause pain. This hypothesis would also explain the marked variability which other observers have found, as well as ourselves, in the relief of pain by sympathetic denervation in interstitial cystitis. If the area of irritation of the bladder wall was in the region of the trigone, then sympathetic denervation would be likely to afford a major degree of relief, whereas, if the focus of irritation were chiefly in the fundus, it would not.

In order to increase the bladder capacity in these patients, it is often desirable to carry out a progressive dilatation of the bladder for several weeks or months after operation. We are convinced that the discomfort associated with such forceful dilatation is more effectively abolished by this more complete form of sympathetic denervation which we have carried out than by the simple removal of the superior hypogastric plexus alone. We have also established the fact that the relief of pain achieved by sympathetic denerva-

tion is not merely a temporary effect. We have observed, in our cases, that it continues for at least three years.

One further step has been added in the effort to produce more complete relief. This arose in connection with Case 6. For technical reasons due to a previous inflammatory episode, it was thought inadvisable to attempt exposure and removal of the right upper sacral chain. Although this patient had experienced some relief of pain after operation, it was quite incomplete. Subarachnoid alcohol injection was then employed, and was found to be very successful in relieving the residual pain. Consequently, this additional step is now used when the patient has persistent pain after sympathetic denervation. Possibly in those cases where the trigonal region is not involved, certain cases of interstitial cystitis, intraspinal alcohol alone would suffice to relieve the patient. This question should be further investigated. Where the trigonal region is chiefly involved, probably sympathetic denervation will be found irreplaceable and the intrathecal injection of alcohol only a subsidiary method. Unusual precautions should be taken in administering the alcohol injections to prevent any injury to the motor nerves of the bladder. This phase is being presented in a subsequent communication.⁸ The subarachnoid injection of alcohol, as used in these cases, probably actually interrupts, in the intraspinal part of the posterior roots, some, at least, of the fibers from the pelvic and internal pudendal nerves carrying the painful impulses from the bladder to the cord.

There is one further step in the clinical investigation of these cases of intractable bladder pain that we are now investigating, namely the results obtained by temporarily interrupting the sympathetic nerves and the pelvic roots separately. Flothow⁹ has suggested and described a relatively simple method for anesthetization of the hypogastric nerves. A low spinal anesthesia, on the other hand, will block the sacral nerves while sympathetic pathways which reach the spinal cord at a much higher level will be intact. By studying the effect on the pain of these two procedures as a preoperative test, it is our belief that, in the future, we can intelligently select the type or types of denervation that will relieve the intractable pain of tuberculosis of the bladder and interstitial cystitis.

CASE REPORTS

Case 1.—No. 90961: M. D., female, age 12. C.C.: Frequency of urination. P.I.: Six years ago, patient first noted intermittent attacks of frequency of urination and dysuria. Four years ago, acid-fast bacilli were identified in the urine from each kidney. Frequency gradually increased to every half hour and incontinence at night. Treated at sanatorium four years. Unable to attend school. P.H.: Scarlet fever at age 8, without complications. F.H.: No tuberculosis or known contacts.

P.E.: Undernourished girl, age 12 appearing chronically ill. Teeth: Two carious. Tonsils large and cryptic. Neck: Small, shotty nodes. Lungs: Clear to auscultation and percussion. Heart: Soft systolic murmur at apex. B.P.: 148/80. Abd.: No masses or tenderness and no C.V.-angle tenderness posteriorly. Lab.: Urine: Alb. 1 plus; 1-2 W.B.C. per H.P.F.; no R.B.C.; acid-fast stain negative. Guinea-pig positive for tuber-

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culosis. P.S.P., 35 per cent first half hour, 60 per cent in two hours. Urea clearance: First hour C.M. 85 per cent; second hour C.M. 79 per cent.

Cystoscopic examination, under caudal anesthesia, with bilateral pyelograms: Showed bilateral calcium deposits in both kidney regions with deformity of calcine structure. Indicative of bilateral renal tuberculosis. Bladder capacity 80 cc. Frequency every half hour.

Operation.—July 3, 1934: A superior hypogastric plexectomy and exeresis of the sympathetic chain from the first to third sacral ganglion was performed.

Postoperative Course.—Wound healed by primary intention. On the tenth P.O. day, bladder capacity 150 cc. and frequency reduced from every 15 to 30 minutes to one to one and one-quarter hours. Followed in outpatient department by weekly bladder dilatation and gomenol instillations. After 12 weeks, was able to attend school for the first time in four years. No enuresis. Able to go two hours between voidings. Five months after the operation, frequency of every two hours; bladder capacity 180 cc.; no pain whatsoever.

Case 2.—No. 69243: A. F., male, age 26. C.C.: Frequency and dysuria. P.I.: Three years before admission began to notice increasing dysuria, frequency and nocturia every one-half to one hour. Associated with suprapubic discomfort and cloudy urine. P.H.: Noncontributory. F.H.: Negative for tuberculosis or contacts.

P.E.: Young adult male, undernourished and appearing chronically ill. Neck: No enlarged nodes or scars. Lungs: Negative to auscultation and percussion. Abd.: No C.V.-angle or kidney tenderness anterior. Tender over suprapubic area. Genitalia: Epididymis and vasa normal. Rectal: Prostate indurated in both lateral lobes, also seminal vesicles. Lab.: Blood studies not abnormal. Wassermann negative. N.P.N., 35. Urine: Alb. 1 plus, acid, cloudy, many W.B.C. and acid-fast bacilli. P.S.P., 77 per cent in two hours.

Intravenous pyelography showed a destructive lesion in both kidneys. At cystoscopy, the left ureter only could be catheterized. Positive for acid-fast bacilli. Guinea-pig inoculation was positive. Discharged to sanatorium for treatment. *Diagnosis:* Bilateral tuberculosis of the kidneys.

The patient was readmitted to the hospital in October, 1934, two years later, at which time the bladder capacity was 90 cc.; frequency every half hour, accompanied by severe, burning dysuria. Had lost weight and strength and was going down hill. Cystoscopic examination showed the bladder to be markedly inflamed, with golf hole ureteral orifices on both sides. No attempt made to catheterize the ureters.

Operation.—October 5, 1934: Resection of the superior hypogastric plexus and exeresis of the right sacral sympathetic chain from the first to third sacral ganglion was performed.

Postoperative Course.—Relief of pain on voiding and suprapubic discomfort. Bladder capacity raised to 175 cc., voluntarily. Patient was given bladder dilatation and instillations. Further dilatation than 155 cc. with irrigator caused severe renal colic on right side. (Dilatations similar to those employed by Bumpus in treatment of interstitial cystitis, i.e., with irrigator four feet above patient, giving approximately 120 Mm. mercury hydrostatic pressure.)

Eight months after operation, goes as long as two hours between voidings. Nocturia four times. No bladder or suprapubic discomfort. Occasionally slight terminal dysuria referred to urethra. Has gained in weight and strength; able to work as apprentice printer.

Case 3.—No. 81804: J. C., female, age 18. C.C.: Frequent and painful urination. P.I.: Fifteen months before admission, patient began to notice painful, frequent urination which gradually increased up to incontinence, especially at night. Three months after onset noted radiating pain from right C.V.-angle to groin. Was cystoscoped at another hospital and a diagnosis of bilateral tuberculosis of the kidneys was made, confirmed by guinea-pig inoculation, and the patient was sent to a sanatorium for treatment, where

frequency increased to every 15 minutes. Placed on constant catheter drainage for eight months. P.H.: Six years previously, loss of weight and strength, no diagnosis made. Four years ago pain in right shoulder, which later became ankylosed, without drainage. F.H.: No tuberculosis or contacts.

P.E.: Undernourished girl, age 18, appearing to be in continual distress from bladder pain. Right shoulder ankylosed at the humeroscapular joint. Lungs: Right apex fine crepitant râles. Abd.: Scaphoid; right kidney lower pole tender on palpation. Right C.V.-angle tenderness. Lab.: R.B.C. 4,900,000; W.B.C. 12,000; Hb. 90 per cent, P.S.P., 35 per cent in two hours. Urine: Alb. and sugar negative, rare W.B.C. and R.B.C., many bacilli (gram-negative). Acid-fast bacilli present. Guinea-pig positive. Wassermann negative.

When first seen, the patient was leading a catheter life. Bladder capacity could be raised to 75 cc., which, however, caused severe pain to develop in the right C.V.-angle, accompanied by chills and fever following each bladder dilatation attempted.

Operation.—November 13, 1934: Resection of superior hypogastric plexus and exeresis of the sympathetic chain, bilaterally, from the first to the third sacral ganglion, was performed.

Postoperative Course.—Incontinent for first three days, but had complete relief from pain. In six days, was voiding every three-quarters to one hour. In one month, bladder capacity 125 cc. and patient going up to two hours between voidings. Nocturia four times. In two and one-half months, bladder capacity was 290 cc. and continues between 275 to 300 cc. since (Chart 1).

Case 4.—No. 12743: R. B., male, age 42. C.C.: Painful frequent urination. P.I.: Seven years before admission, patient first noted nocturia followed by hematuria. Two months later, swollen left epididymis, which broke down, drained and healed slowly. Following this he developed severe dysuria and frequency. Right epididymitis developed two months before admission, with epididymectomy elsewhere. Symptoms increased, so that he was voiding every ten to 15 minutes, day and night, with severe dysuria. No relief by bladder irrigations and instillations. P.H.: 1911, pleurisy with effusion. 1912, tuberculosis right hip with draining sinuses and ankylosis. 1927, Pott's disease with psoas abscess. 1928, ischiorectal abscess; left tuberculous epididymitis. 1930, tuberculosis left hip with ankylosis.

P.E.: Undernourished male, age 42, appearing chronically ill. Both hips were ankylosed in an extended position. In continual distress from pain and frequent urination. Numerous scars and draining sinuses over hips, genitalia and lower extremities. Teeth: Carious. Lungs: Negative on repeated examinations. B.P. 118/62. Heart: Normal. Abd.: Tip of spleen felt. No C.V.-angle tenderness. Moderately severe suprapubic tenderness. Genitalia: Draining sinuses from epididymies. Vasa indurated and nodular. Rectal: Prostate firm and irregular. Lab.: Urine: Alb. 2 plus; 15–20 W.B.C. per H.P.F., no R.B.C. Guinea-pig positive for tuberculosis. N.P.N. 40, R.B.C. 3,500,000, W.B.C. 12,350. Cystoscopy enabled one to see only vault of bladder because of ankylosis of hips and spine. Intravenous pyelograms showed evidence of a destructive lesion in both kidneys.

Operation.—November 21, 1934: Resection of the superior hypogastric plexus and exeresis of the sacral sympathetic chain from the first to the third ganglion was performed.

Postoperative Course.—Continued to void frequently for two days, but had no pain. Seven days postoperative, wound broke down, requiring secondary closure. In three weeks, patient was going as long as two and one-half hours between voidings with no dysuria, bladder capacity 175 cc. At eight months, he was voiding every two and one-half to three hours, without pain. He has had no dilatations or bladder treatments since operation.

Case 5.—Hosp. No. 77573: A. B., male, age 48. C.C.: Frequent painful urination. P.I.: Began four years before admission with gradually increasing frequency of urina-

tion and dysuria, especially terminal. Ten months before admission, had frank hematuria, and following this, became symptomatically much worse. Voided as frequently as every three to ten minutes, day and night, with severe pain, and developed bilateral tuberculous epididymitis. Treated at a Veterans' Hospital for three months with usual measures without relief. Then treated in the outpatient department with bladder irrigations with various drugs, bladder dilatations and bladder instillations of gomenol, following which he voided less frequently, but pain continued. P.H.: Twenty years ago, had pleurisy with effusion. Nine years ago, had frank hemoptysis; treated with sanatorium regimen and tuberculosis apparently arrested.

P. E.: Undernourished white male. Teeth: In poor state of repair. Voice: Slightly husky. Lungs: Dulness in both apices with fine râles in right apex, both anterior and posterior. Abd.: Neither kidney felt or tender. Suprapubic tenderness. Rectal: Tone good. Bilateral indurated epididymis both very sensitive. Prostate slightly enlarged, firm and irregular with induration of seminal vesicles, suggesting bilateral tuberculous seminal vesiculitis and prostatitis. Lab.: Acid-fast bacilli from both kidneys. Typical tuberculous lesions found in microscopic sections from removed epididymies. At cystoscopy, under caudal anesthesia, the bladder capacity was raised to 125 cc. Bilateral pyelograms (retrograde) showed deformities typical of tuberculosis, which was confirmed by guinea-pig inoculation.

Operation.—March 23, 1935: Presacral neurectomy and exeresis of both sacral sympathetic chains including one to two sacral ganglia was performed.

Postoperative Course.—During the first few days, voided small amounts frequently, then capacity rose to 275 cc. on third postoperative day, and went two to three hours between voidings, which improvement has continued. Patient has no pain or dysuria. In the outpatient department he is continuing with bladder dilatations and instillations at regular intervals to keep his bladder capacity up.

Case 6.—No. 2178 J: A. G., female, age 26. C.C.: Painful frequent urination. P.I.: First seen in February, 1934. Six months previously began to notice frequency every hour and nocturia every two hours. Voided about 60 cc. at a time. There was associated pain in the right groin which radiated to the urethral meatus. Marked dysuria has been present since onset. F.H.: Three relatives died with tuberculosis. No known contacts. *Diagnosis:* Tuberculosis in the right kidney. A nephrectomy and ureterectomy was performed in March, 1934. Frequency persisted in spite of the nephrectomy, and general and local measures toward treatment of the cystitis. A hysterectomy was performed in January, 1935, for fibroid uterus. Bladder symptoms persisted; the patient was voiding every five to 15 minutes with considerable pain.

Operation.—December 2, 1935: Presacral neurectomy and a unilateral (left) exeresis of the sacral sympathetic chain was performed. It was impossible to attack the right ganglia because of adhesions of the peritoneum and stump of the uterus.

Postoperative Course.—Within six days, frequency was diminished to every two to three hours, and within 13 days the bladder capacity could be raised to 300 cc., although some pain persisted and there were periods in which the frequency would recur. Subarachnoid alcohol was undertaken with the patient lying on her left side, in April, 1936, using 0.75 cc. of absolute alcohol. This was repeated with the patient lying on the opposite side, in June, 1936, since which time, she has had complete relief of pain in her bladder, dysuria, and frequency. She voids now every three to four hours and nocturia two times (which has persisted up until the present time).

Case 7.—No. 2906-J: R. M., female, age 32. C.C.: Painful frequent urination. Pain in urethra on walking. P.I.: Patient had symptoms of tuberculous cystitis with frequency and dysuria seven years ago. Nephrectomy performed, with relief of all bladder symptoms until nine months ago, then noted gradually increasing frequency and nocturia with associated dysuria, and for the last two months noted marked frequency every 15 to 20 minutes and urethral pain on walking. P. H.: Also, had pulmonary tuberculosis and apparent arrest in process by collapse therapy.

P. E.: Young, adult female, appears chronically ill but fairly well nourished. Lungs: Thickened pleura but no active lesions. Abd.: Scar of nephrectomy on left. No palpable organs or masses. Right kidney region not tender. Pelvic: Urethral orifice red and pouting. Entire urethra tender on pressure. Lab.: Acid-fast bacilli in urine from remaining kidney. Secondary infection with bacilli (many). The patient was treated by all types of irrigation, instillation and mouth medication with symptoms increasing for nine months. Cystoscopy revealed urethritis, cystitis with ulcers near both ureteral orifices and right lateral wall of bladder.

Operation.—September 30, 1937: Presacral neurectomy and exeresis of lateral sacral sympathetic chain from first to third ganglion, bilaterally was performed.

Postoperative Course.—Relief of pain except in urethra. Frequency every 30 to 45 minutes. Incontinent for first two days, then occasionally nocturnal enuresis for two weeks. Three months later secondary infection became marked, accompanied by severe urethral burning on walking, with frequency at times half hourly, but could go up to two hours while at rest. Intrathecal alcohol injection, February 8, 1938, with right side uppermost, almost entire relief of pain with frequency diminished to three hours. February 14, 1938, intrathecal alcohol injection with left side uppermost. Complete relief of pain. Frequency every three to four hours, day and night. Walks without pain in urethra.

Case 8.—Hosp. No. 25163: R. C., female, age 44, was originally admitted to Iola Tuberculosis Sanatorium, in 1929, for minimal pulmonary tuberculosis. In 1933, she had tuberculosis of the left hip for which she was twice fused, in 1933, and again in 1934. She was readmitted to Iola Sanatorium in January, 1937, for reactivation of pulmonary tuberculosis; and shortly thereafter developed tuberculosis of the left sacro-iliac joint, for which a fusion was performed. Shortly thereafter, she developed frequency and burning, hematuria and pyuria. Tubercle bacilli were demonstrated in the urine. In spite of conservative measures, the bladder pain became increasingly severe, and wearing on the patient, causing her to lose much sleep.

Operation.—September 9, 1937: The superior hypogastric plexus and the right upper sacral sympathetic chain were excised. The left sacral chain was so embedded in scar tissue from the sacro-iliac disease that it was considered inadvisable to attempt its removal. The patient was transferred back to Iola Sanatorium on the eleventh post-operative day, markedly relieved of her bladder pain. If her symptoms recur (associated with the incompleteness of the operation), it is planned to reinforce the operative treatment with subarachnoid alcohol injection.

Case 9.—G. P., male, young adult. This was a patient seen in consultation with Dr. Elmer Belt, at the Good Samaritan Hospital, Los Angeles, Calif. He had the typical findings of an interstitial cystitis of severe grade. In spite of vigorous conservative measures, the bladder pain, tenesmus and frequency continued without relief. The patient in the previous six months had lost 25 pounds in weight from the constant pain and loss of sleep. He had to void every ten to 15 minutes, day and night. Doctor Belt was planning to give him relief by transplanting his ureter into the colon.

Operation.—The superior hypogastric plexus and the upper sacral sympathetic chains were removed July 7, 1935.

Postoperative Course.—Six weeks later he had regained 20 pounds in weight; was voiding every three or four hours; his bladder capacity was 300 cc., and his only annoying sensation was a burning in the urethra during urination. His bladder pain apparently had been completely eliminated and his general health was excellent. This improvement continued until he was lost track of about a year after operation.

Case 10.—Hosp. No. 94259: E. H., female, age 23. This patient presented a typical example of interstitial cystitis. She had been in the hospital on seven previous occasions, during the past three years. She had had numerous courses of irrigations, instillations, fulgurations and dilatations of the bladder without any lasting effect on the severe pain, tenesmus, nocturia and frequency. Her bladder capacity was 80 to 90 cc. and she voided every 15 to 30 minutes. Urine culture had shown *Staphylococcus albus*, *B. coli*,

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B. proteus, *B. aerogenes* and *B. alkaligenes* on various occasions but no tubercle bacilli on guinea-pig inoculation.

Operation.—The superior hypogastric plexus and the upper sacral sympathetic chain on each side were removed June 5, 1937.

Postoperative Course.—Within two weeks her bladder capacity had increased to 200 cc. and her symptoms were much improved. However, within six months her dysuria and frequency had returned, with a highly infected urine, and it was felt that the sympathetic denervation had not helped her very appreciably.

Case 11.—Hosp. No. 131876: E. C., male, age 54. For over two years this patient had had frequency, urgency, burning and nocturia. The pain on urination had increased. Various conservative measures had failed to relieve it. Cystoscopy showed numerous bleeding areas along the right and left walls and over the fundus of the bladder. Tubercle bacilli were absent from the urine.

Operation.—October 20, 1937: The superior hypogastric plexus and both upper sacral sympathetic chains were removed.

Postoperative Course.—The patient obtained a considerable degree of relief. The frequency and pain were much less, the bladder capacity increased from 100 to 275 cc. with the aid of dilatations. At present he still has some bladder pain but much less than before operation, and he now sleeps well at night. We have offered him sub-arachnoid alcohol injection but he does not feel that the pain is sufficiently troublesome to warrant coming in to the hospital for this at present.

REFERENCES

- ¹ Pieri, G.: (a) Enervation or Ramisection? *Presse méd.*, **34**, 1141, September 8, 1926.
(b) Contributi clinici alla chirurgia del sistema nervoso vegetativo: la cura della nevrite ascendente. *Arch. ital. di Chir.*, **27**, 288, 1930.
- ² Learmonth, J. R., and Braasch, W. F.: Resection of Presacral Nerve for Disease of Bladder. *Tr. Am. A. Genito-Urin. Surgeons*, **25**, 313, 1932.
- ³ Douglass, H. L.: Excision of Superior Hypogastric Plexus of Intractable Interstitial Cystitis. *Am. J. Surg.*, **25**, 249, 1934.
- ⁴ Learmonth, J. R.: Value of Neurosurgery in Certain Vesical Conditions. *J.A.M.A.*, **98**, 632, February 20, 1932.
- ⁵ Learmonth, J. R.: Contribution to Neurophysiology of Urinary Bladder in Man. *Brain*, **54**, 147, 1931.
- ⁶ Cheetham, J. G.: Presacral Neurectomy for Relief of Certain Types of Bladder Dysfunction and Pain. *J. Urol.*, **37**, 148, 1937.
- ⁷ Learmonth, J. R.: Surgery of Sympathetic Nervous System. *Brit. J. Surg.*, **25**, 426, October, 1937.
- ⁸ Schroeder, C. F., and Cumming, R. E.: Paper to be presented before the Section on Urology of the American Medical Association, June 15, 1938.
- ⁹ Flothow, P. G.: Relief of Pelvic Pain. *South. Surg.*, **4**, 207, June, 1935.

DISCUSSION.—**DR. LOYAL DAVIS** (Chicago, Ill.): Mr. President and Gentlemen: I think that Doctor Scott's paper and his work offer a very excellent example of the clinical application of known physiologic and anatomic facts in a field which is very difficult to investigate from a purely experimental standpoint through the employment of animal investigation.

We have been interested for a considerable period of time in the pathway of pain impulses, particularly from the viscera, and our chief concern has been to try to work out the mechanism for the pathway of these visceral afferent impulses.

As Doctor Scott has said, he has been unable to give a conclusive answer to the question of whether or not the painful impulses are the result of the action of the efferent mechanism of the sympathetic fibers, which produces

contraction of smooth muscle, in itself is painful, and which are then carried by the ordinary somatic nerves to consciousness, or whether the visceral painful impulses are transmitted by an afferent mechanism over the sympathetic fibers.

As he has told you, the innervation of the bladder is an extremely complicated one, and, as he has pointed out, there are various regions of the bladder which are innervated by sympathetic fibers coming from the thoracic lumbar segment of the spinal cord, and other portions of the bladder which are innervated by fibers coming from the sacral portion of the cord, in other words, parasympathetic fibers.

Both of these types of fibers contain small myelinated and unmyelinated fibers which could carry afferent impulses. In other words, both the pelvic nerves and the hypogastric nerves to the bladder are both motor and sensory in function. Although Doctor Scott has unquestionably obtained clinical relief in his cases, we are not much nearer to the solution of the exact mechanism resulting in visceral pain, because in removing the hypogastric supply to the bladder, both efferent and afferent fibers have been sectioned.

I think it is in this type of work—the clinical application of known physiologic and anatomic facts—which will finally bring solution of the mechanism of the relief of pain. I feel very strongly that the section of sympathetic fibers for the relief of vesical pain is not as effective as the interruption of the pathway, either within the spinal cord or before the fibers get to the spinal cord in the dorsal roots, because I think the mechanism is an efferent and not an afferent one.

In performing a chordotomy, for the relief of visceral pain, it is necessary to make a deep section within the spinal cord so that a portion of the gray matter of the cord is interrupted, because visceral fibers which carry pain go into the spinal cord and then ascend in the cord within the gray matter and not in the white matter, as does pain from the periphery.

THE REPAIR OF ABDOMINAL INCISIONS

ALLEN O. WHIPPLE, M.D., AND ROBERT H. E. ELLIOTT, JR., M.D.

NEW YORK CITY, N. Y.

FROM THE DEPARTMENT OF SURGERY, COLUMBIA UNIVERSITY, NEW YORK CITY, N. Y.

WOUND repair and wound healing are a constant problem in surgery. The surgeon makes incisions in the abdomen more than in any other region. He should be concerned with the best methods of repairing these wounds. Every thoughtful surgeon is interested in the processes that insure the optimum healing and restoration of the abdominal wall to as nearly normal as possible.

With all the importance of this subject and the general interest of surgeons in their daily dealing with it, there is an astonishing dearth of accurate description of the technic of abdominal wound closure as compared with the repair of other wounds, especially of a plastic nature. William S. Halsted,¹ in his epoch making paper in 1913, in which he presented the silk technic and its essential philosophy which he had developed at the Johns Hopkins Hospital, devotes only a short paragraph to the discussion of abdominal wound repair, but gives no details as to type of suture and method employed.

In 1932, Dr. Arthur Dean Bevan² presented a paper before the American Surgical Association entitled "Abdominal Incisions and Their Closure." In this paper special stress was laid on the description of various incisions, and emphasis was placed on the closure, giving the details of a technic worked out by himself at the Presbyterian Hospital, in which retention sutures of silkworm gut were tied over pearl buttons. No definite figures were quoted, however, relative to the percentage of wound disruption and of postoperative ventral hernia following closure of these various incisions, but in other respects this is a most comprehensive discussion of the subject of abdominal incisions.

It is only in recent years that the frank and honest discussion of wound infection and abdominal wound disruption, as a result of the study of carefully recorded and analyzed hospital records in our best hospitals, has revived an interest in wound healing.

The repair of abdominal incisions presents particular problems that are not met with in other wounds or other regions. These may be analyzed under the following headings:

(1) The peculiar arrangement of the flexing and rotating muscles, and the aponeurotic layers entering into the complex functions of the muscles of the abdominal wall (Figs. 1, 2, and 3). The lateral pull of the oblique and transversalis muscles on the outer edges of vertical incisions is an almost daily, distressing observation in the closure of such wounds.

(2) The repaired abdominal incisions, especially those in the upper abdomen, are subject to peculiar stress and strain as a result of vomiting, coughing, hiccough, distention and the lifting and moving of the patient by the

attendants in the administrations consequent to the many daily physiologic demands and in nursing care. The sudden pain associated with the above factors increases the lateral pull of the rotating muscles, and increases intra-abdominal tension.

(3) The abdominal incisions are more frequently contaminated with virulent and necrotizing aerobic and anaerobic organisms than any others.

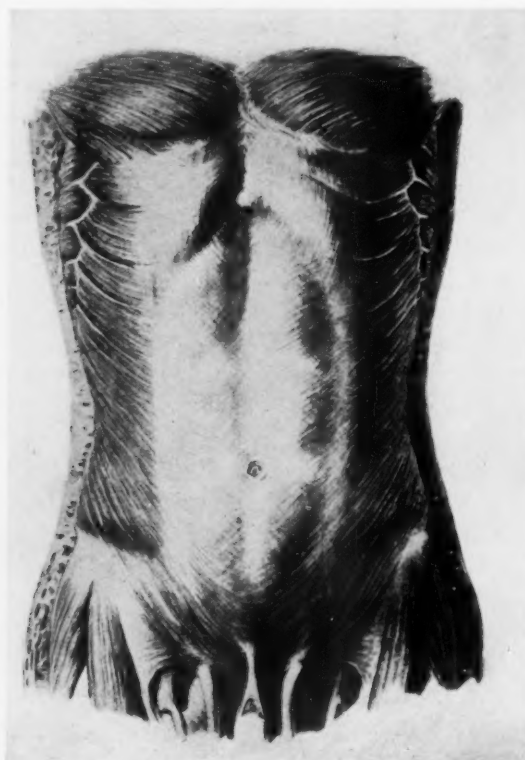


FIG. 1.—To show direction of muscle and aponeurotic fibers of superficial abdominal muscles. (From Spalteholz, 2nd edition.)

Activated enzymes are at times in contact with drained incisions in patients requiring intestinal repair, followed by fistulae. These are factors which not only inhibit normal wound healing but may actually digest the tissues and dissolve the absorbable catgut sutures used in the repair. In such wounds the factors of increased intra-abdominal tension are most often associated, and it is in these patients that wound disruption and ventral hernia are most frequently seen.

(4) In many elderly or cachectic patients suffering from prolonged malnutrition and vitamin deficiencies, and requiring extensive operations for resections of malignant growths of the gastro-intestinal tract, the low serum protein content of the blood and the tissues prevents normal healing and unquestionably predisposes to wound disruption:

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(5) Because of the dread of the factors mentioned under the above headings, many surgeons believe that heavy suture material should be employed both in the layer repair and in the tension or reinforcing sutures. Heavy chromic catgut, in double strands, as high as No. 2 grade, is used in many clinics. One has but to watch the inexperienced house surgeon or assistant pull up on these continuous sutures, or the heavy silkworm gut or metal re-



FIG. 2.—To show direction of muscle and aponeurotic fibers of deeper abdominal layers. (From Spalteholz, 2nd edition.)

inforcing sutures, to appreciate the amount of tissue necrosis that will inevitably result within the following 24 hours. A study of microscopic sections in wounds so repaired reveals long transverse lines of tissue necrosis on either side of the repaired incision (Figs. 4 and 5). The necrosis takes place until the tension between suture and tissue is relieved. This tension, with a running continuous tight suture, diminishes the blood supply to the very tissues in which the surgeon is attempting to encourage wound healing. This mistake of tight suturing is probably the most common one made in the repair of abdominal incisions.

Because of the fear of wound infection, and persistent sinus formation resulting from employment of nonabsorbable sutures, catgut is used by the

majority of surgeons in abdominal work. In wounds contaminated with lower ileal and colon contents, nonabsorbable sutures should not be used; but in such wounds, contaminated with necrotizing organisms, and in incisions where activated pancreatic ferments are apt to be secreted, as in duodenal, jejunal and pancreatic operations, catgut has very definite drawbacks. The irregular and early absorption of both plain and chromic catgut sutures in



FIG. 3.—To show direction and distribution of nerves to abdominal wall. (From Spalteholz, 2nd edition.)

the presence of intestinal ferments has been noted by every experienced surgeon and has been experimentally demonstrated.³ Another factor, which has only within recent years been pointed out,^{4, 5} is the allergic reaction of catgut in patients showing edema of the wound edges and in disruption of abdominal wounds. Kraissl,⁴ working in our Surgical Laboratory, sensitized 52 guinea-pigs to plain and chromic catgut. Celiotomies were performed upon these animals. Thirty per cent of these guinea-pigs disrupted their abdominal wounds. All of a series of 26 control guinea-pigs healed normally except one. There is little doubt but that the local reaction in patients allergic to catgut predisposes to infection and favors wound disruption.

REPAIR OF ABDOMINAL INCISIONS

Contaminated Incisions With and Without Abscess.—In abdominal incisions contaminated with ileal and colon contents our present technic is as follows: If an abscess, as in appendicitis, is present, it is drained with one or



FIG. 4.—(Two day wound.) There is a moderate amount of cellular infiltration around the catgut sutures, thick, swollen strands of which are seen to the left surrounded by exudate. On the right side are seen the silk sutures surrounded by practically no exudate. Fibroblasts and granulation tissue are already evident around both catgut and silk sutures. Repair has already started.

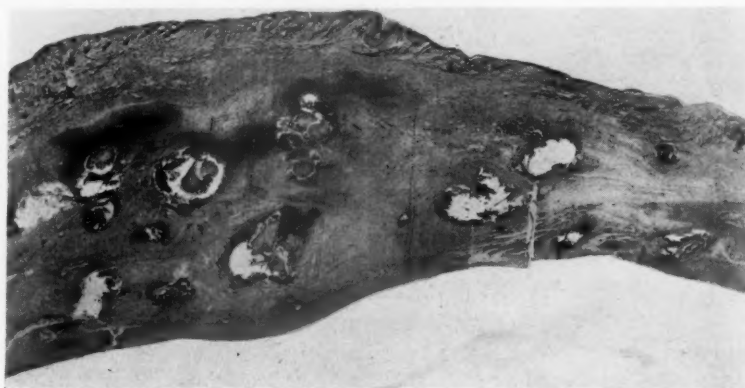


FIG. 5.—(Six day wound, catgut on the left side, silk on the right side.) All silk fibers are separated by an ingrowth of fibroblasts and giant cells. In striking contrast, there is no growth immediately around the catgut, which is surrounded first by a pool of exudate, then degenerated muscle, and then by granulation tissue on the outside of this. This is a very striking contrast. Note the difference in the thickness of the wall on the catgut and silk side, due to excessive edema of the tissues where catgut was used.

two soft rubber tubes or cigarette drains, introduced through a small opening in a China silk tampon, the peritoneum is closed with interrupted No. 00

chromic catgut sutures around the drains, and the wound is then packed with weak iodoform or zinc peroxide gauze around the drains inside the silk tampon. No attempt is made to suture the muscles, subcutaneous tissues or skin. The patient must be kept in bed longer than the ones with sutured wounds, until the wound has filled in with granulation tissue.

If an abscess is not present but the wound edges are contaminated with ileal or colon contents, as in an open resection, a small Penrose drain is placed near the site of repaired intestine, the peritoneum is closed about it, and at least the central part of the wound is tamponed as in the case of the abscess.

Clean Abdominal Wound Repair.—We tend, more and more, to employ fine silk in all abdominal work. Frequently the hemostats are left on until the lesion in the abdomen is revealed, when either catgut or silk can be decided upon. They should not be used together, as it has been demonstrated⁶ that catgut favors the growth of bacteria in the wound; and in an infected wound, silk, unless in very fine grades, is more apt to act as a foreign body and cause protracted sinuses. The only abdominal wounds not associated with abscesses that we drain now are resections for gastric and colon cancers, cholecystectomies, especially where the cystic duct stump is not adequately peritonealized, and common duct drainages.

In upper abdominal operations we determine the type of incision largely by the width of the intercostal angle—using the transverse for the wide-angled, obese patient, and the split rectus for the narrow costal-angled, thin individual. We prefer transverse incisions because the lateral pull of the oblique and transversalis muscles tends to close rather than open the incision. This is graphically demonstrated in patients not thoroughly relaxed under anesthesia. Furthermore, the transversely cut rectus sheaths with their transverse fibers hold the sutures much more securely, and do not tend to tear out.

Because of our previous experience with disruptions, some five years ago we adopted a technic for abdominal wound closure which in our hands has practically eliminated, in our clean cases at least, wound disruption and post-operative hernia. In both transverse and vertical incisions, in both upper and lower abdomen, we have employed and are continuing to employ the following technic for closure: Peritoneum and posterior rectus sheath or trans-

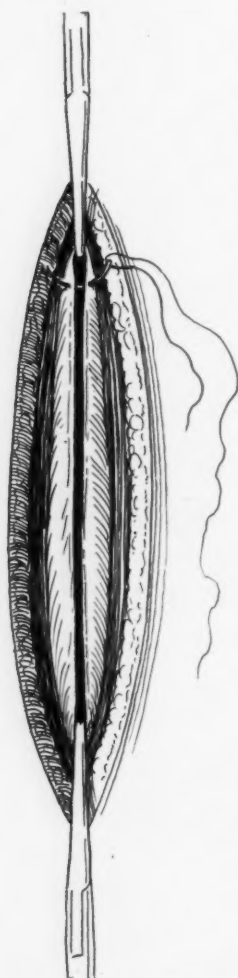


FIG. 6.—Closure of peritoneum and posterior rectus sheath of transversalis fascia.

REPAIR OF ABDOMINAL INCISIONS

versalis fascia, continuous, fine C silk or No. 00 chromic catgut followed at 2 cm. intervals with interrupted sutures. This is done to bring a continuous

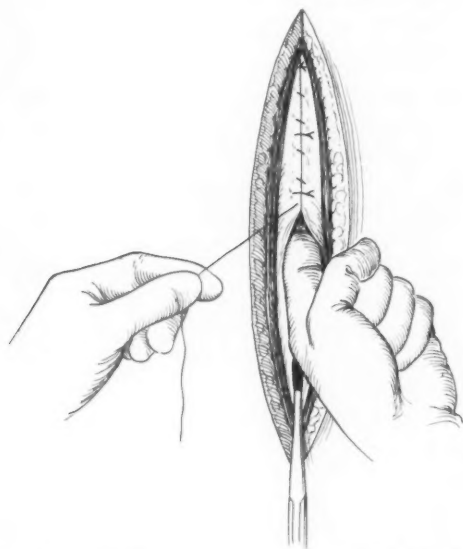


FIG. 7.—Showing the use of continuous fine silk followed by interrupted silk at 2 cm. intervals.

surface of peritoneum to peritoneum, to insure prompt agglutination of peritoneal edges, and to prevent possible projection or protrusion of omental tabs into the peritoneal repair (Figs. 6 and 7). The anterior rectus sheath and oblique muscles (in the transverse incisions) are repaired with the same fine silk or chromic catgut by the use of a vertical figure-of-eight, or what we call the "far-and-near" stitch, at intervals of 7 to 8 Mm. This stitch is begun by introducing the needle 5 Mm. from the edge of one sheath out through the margin of the other edge, into the margin of the first edge and out 5 Mm. from the margin of the opposite sheath. Slight tension on the suture ends approximates the two edges of the sheath. These interrupted sutures should be tied loosely with a square knot, to allow for the take-up that results from the occurrence of the edema of repair, and to prevent any cutting of the sheath by the suture (Fig. 8). This is a tensionless suture, if loosely tied, which prevents tissue necrosis, and therein lies its virtue. Proper hemostasis will obviate any need for subcutaneous sutures. The skin is closed with

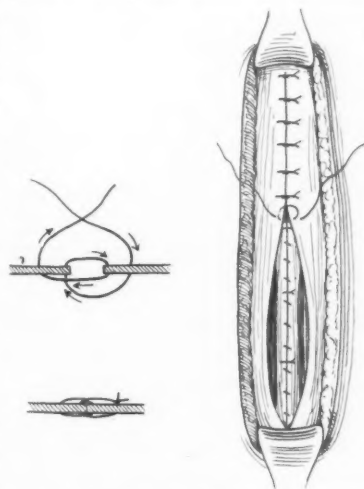


FIG. 8.—Closure of the anterior rectus sheath with "far-and-near" interrupted fine silk sutures.

interrupted silk sutures on separate cambric needles to avoid the contamination of the silk by repeated puncture of hair follicles and sweat glands with the same needle and the same long suture (Fig. 9).

We claim no originality in this technic, although we have not seen this identical procedure described. Babcock⁷ describes this suture as "a combined relaxing and coapting suture, one of the best sutures for use where there is tension." Dr. H. H. Lyle of St. Luke's Hospital informs me that this "far-and-near" suture has been used for many years on his service, employing catgut, and that Thomas Markoe began using it for rapid closure of war wounds during the Civil War. Dr. Daniel F. Jones used a similar stitch for

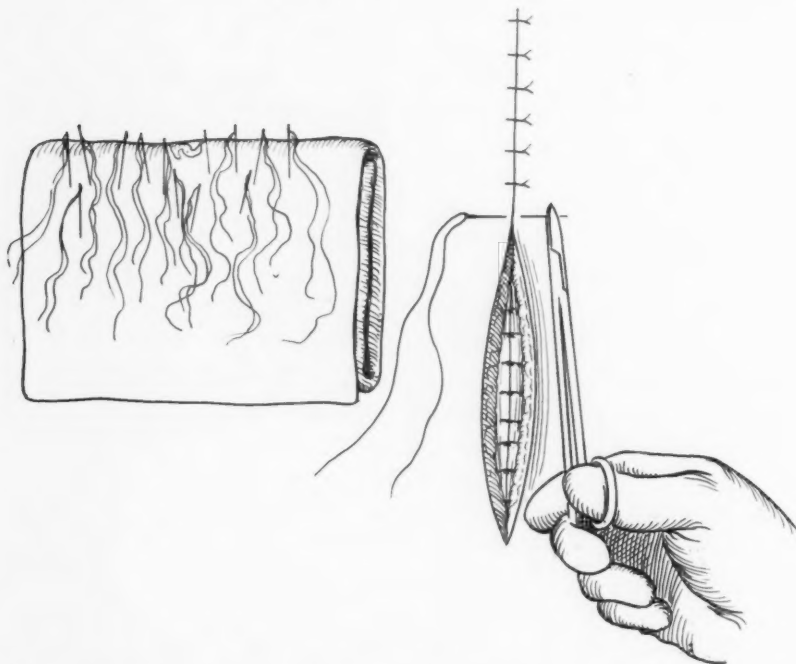


FIG. 9.—Closure of skin and subcutaneous tissue with interrupted fine silk on separate cambric needles.

deep retention sutures. Undoubtedly it has been used by others, as we are using it, but we have failed to find a description of the technic for abdominal wound closure such as we are now employing. We do know it has reduced wound disruption and postoperative incisional hernia to a minimum in our postoperative and follow-up studies. For this reason we have abandoned the use of retention sutures, which seldom proved effective, and frequently caused stitch abscesses.

In a control series of 300 abdominal wounds, with the layers closed with catgut, bolt retention sutures tied over pearl buttons were used in the great majority of cases. We first saw these pearl button retention sutures used in Bevan's Clinic at the Presbyterian Hospital, Chicago. Yet the incidence of

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wound infections, disruptions and postoperative ventral herniae was far greater (Tables I to V). Table VI shows the distribution of cases in the series reported and in the control group. It will be observed that the number of biliary, stomach and bowel cases in the two series are roughly comparable, the numerical difference between the totals of the two groups being due principally to the number of appendicectomies and herniae in the reported series.

TABLE I
THE INCIDENCE OF SUTURE MATERIAL AND THE TYPE OF
SUTURE USED IN THE ANTERIOR SHEATH

Material	No. of Cases	F. & N. Suture	Plain Suture
Silk.....	300	228	72
Catgut.....	181	102	79
Control*.....	300	0	300
Totals.....	781	330	451

* Cases in the control group were sutured with catgut.

TABLE II
INFECTION IN CLEAN OPERATIVE WOUNDS

Material	No. of Cases	Triv. Inf.	Per Cent	Ser. Inf.	Per Cent	Total Inf.	Per Cent
Silk.....	199	4	2.04	0	0.0	4	2.04
Catgut.....	25	1	4.00	0	0.0	1	4.00
Totals...	224	5	2.22	0	0.0	5	2.22
Control.....	53	4	7.55	2	3.77	6	11.32

TABLE III
INCIDENCE OF DISRUPTION

Material	F. & N. Suture	Per Cent	Plain Suture	Per Cent	Total	Per Cent
Silk.....	1	0.44	0	0.0	1	0.33
Catgut.....	4	3.92	1	1.26	5	2.76
Totals.....	5	1.51	1	0.66	6*	1.25
Control.....	—	—	13	4.34	13	4.34

* Two cases have not been included in which dehiscence of the wound took place, because the separation of the wounds only extended down to, but not through, the anterior rectus sheath.

In our tables are included the abdominal incisions repaired, with the technic described, both with fine silk and fine chromic catgut, and a series of 300 cases repaired with chromic catgut in the usual manner, that is, with continuous sutures and reinforced with retention sutures. We use the term trivial infection for any case in which the healing of the wound was not delayed.

This includes single stitch infection or wounds with a serous discharge giving a positive culture. Serious infection is one which delays the normal convalescence and prolongs the bedstay of the patient.

TABLE IV
FOLLOW-UP STATISTICS

Material	Cases Followed	Per Cent Followed	Ave. No. Mos. Followed
Silk.....	173	57.7	13
Catgut.....	111	61.4	17
Totals.....	284	59.2	15
Control.....	203	67.7	52

TABLE V
INCIDENCE OF POSTOPERATIVE HERNIA AMONG
CASES FOLLOWED

Material	No. Herniae	Per Cent
Silk.....	0	0.0
Catgut.....	7	6.31
Totals.....	7	2.46
Control.....	30	14.77

TABLE VI
DISTRIBUTION OF CASES

Type of Operation	Silk	Catgut	Total	Control
Hepatic and biliary.....	81	81	162	140
Stomach and duodenum.....	40	25	65	57
Large and small bowel.....	16	48	64	42
Appendicectomies.....	23	21	44	3
Herniae*.....	52	0	52	7
Miscellaneous.....	88	6	94	51
Totals.....	300	181	481	300

* Including ventral herniae.

Regular staff conferences and well organized follow-up clinics, in our best surgical services all over this country, have made the surgeons, especially the younger group, very critical of any statements regarding wound healing and postoperative herniae based upon impressions and not backed up by searching analyses of carefully kept records. Such studies have revealed a much higher incidence of disruption and ventral hernia in surgical services than the estimates based upon impressions.

Before closing I wish to emphasize again that if silk is to be employed in the repair of wounds, it must be used in the very fine grades. This connotes the minimal tissue damage by the use of sharp knife dissection, fine

hemostats, fine needles, complete hemostasis, absence of tight sutures, careful isolation of skin edges, and protection of exposed tissues from drying and air contamination.

The number of recognized surgeons throughout the country who have adopted silk technic within the past five years is remarkable and speaks for their open-mindedness and their interest in ideal wound healing. It is regrettable that Halsted, who did so much to demonstrate the principles of wound healing, did not live to see the present renaissance of his philosophy.

REFERENCES

- ¹ Halsted, William S.: The Employment of Fine Silk in Preference to Catgut and the Advantages of Transfixing Tissues and Vessels in Controlling Haemorrhage. *J.A.M.A.*, **60**, 1119-1126, 1913.
- ² Bevan, Arthur Dean: Abdominal Incisions and Their Closure. *ANNALS OF SURGERY*, **94**, 555-574, October, 1932.
- ³ Kraissl, Cornelius J.: Intrinsic Factors Altering Absorption of Catgut. *Surg., Gynec. and Obstet.*, **63**, 561-569, 1936.
- ⁴ Kraissl, C. J., Kasten, B. M., and Cimiotti, J. G.: The Relation of Catgut Sensitivity to Wound Healing. *Surg., Gynec. and Obstet.*, **66**, 628-635, 1938.
- ⁵ Jenkins, H. P.: A Clinical Study of Catgut in Relation to Abdominal Wound Disruption. *Surg., Gynec. and Obstet.*, **64**, 648-662, 1937.
- ⁶ Vivier, Peter J.: The Tensile Strength, Gross and Microscopic Morphology and Bacteriology of Celiotomy Wounds Sutured with Catgut and Silk. (Paper to be published.)
- ⁷ Babcock, W. Wayne: A Text-Book of Surgery. W. B. Saunders Company, 606, 1928.

DISCUSSION.—DR. SAMUEL C. HARVEY (New Haven, Conn.): I am very happy indeed to have the opportunity of discussing Doctor Whipple's excellent paper. I shall not go into technical details but I wish to restate the problem in a more general way.

There are two ways, of course, of determining the certitude with which the healing of a wound may take place. One is by experimental investigation upon animals, and the other is actual surgical practice. Both of these are essential for our knowledge of the healing of wounds.

From the experimental work upon animals, we have learned that the healing of wounds follows a normal process. It is a biologic phenomenon, which corresponds to the laws of growth in all biologic things.

We have also learned that there are certain things which interfere with the normal healing process. The process is, in the first place, one of cleaning up tissues which have been devitalized—a matter of three or four days. The secondary part of the process, and the most important, perhaps, is the restitution of the tensile strength of the wound by the growth of the fibroplastic cells.

Theoretically, such a process should take place to the best advantage when there is the least amount of destruction of the cells, when there is no infection, and when there is no foreign material introduced in the wound. Frankly speaking, we have to compromise, because a wound has to be carefully approximated in order to obtain healing. This means that we have to use sutures.

The use of sutures introduces foreign material into a wound. It is just as much the introduction of foreign material as is the devitalization of the cell, the introduction of infection, or the forming of hematomata and serous

accumulations. Therefore, it must be reduced to the absolute minimum in respect to the type of material and in respect to the quantity necessary to achieve the purpose of obtaining approximation of the wound.

Experimentally, that means the reduction of the suture material in size to a minimum and the use of suture material which has the least irritative effect in the wound. Clinically, the same thing follows from studies of the complications of wound healing.

I would like to point out more emphatically than Doctor Whipple perhaps did that no one is really in a position to evaluate the healing of a wound clinically unless he has very carefully, and at the time, personally studied the healing of his own wounds. The impression which all of us may have, that our wounds heal all, or practically all, without any complications, is, I am convinced, a completely erroneous one. A careful follow-up and careful evaluation will show, I think, that 4 or 5 per cent, as a minimum, do not heal as perfectly clean wounds.

A measure of that, perhaps the most characteristic and vital measure, is the disruption of the wound. Again, if one studies very carefully his cases as they come along, he will find a very definite incidence of disruption. I do not think there is any exception to that.

Perhaps more important, with a follow-up he will find that there is a definite percentage of postoperative herniae, and a postoperative hernia is, I think, almost without exception, a disruption of the wound, in most instances not involving the skin but the underlying structures.

Therefore, it seems to me that Doctor Whipple's experience here is a very pertinent one and very much to the point because he has carefully carried over into the clinic the indications resulting from his experimental work.

By the criteria of a careful follow-up and study of his wounds, and with the actual decrease of the complications of healing obtained by the use of a nonirritative suture material, in fine and restricted quantities, with the necessary delicate technic that is required, he has actually demonstrated a very significant decrease in the complications of wound healing.

I should like to add this one thing, however, for those of you who wish to follow this procedure: you should go back to Halsted's article, in 1913, in which he gave the details absolutely essential for the successful carrying out of this technic.

DR. ALBERT O. SINGLETON (Galveston, Tex.): Doctor Whipple is to be congratulated upon his management of this very troublesome vertical, upper abdominal incision. I do not know of anyone who has succeeded so well with it. There is no controversy that I can see about his technic and the reason for his employing it. We have approached this subject from a little different angle and have tried to use a more anatomic incision, more or less upon the principle of the incision advocated some years ago by Sloan.¹

The chief strain or tension upon the abdominal wall, in the upper abdomen particularly, is in a transverse direction, due to the action of the lateral abdominal muscles. The chief holding material of the abdominal wall is the posterior sheath of the rectus muscle, which is the tendinous continuation of the internal oblique and transversus muscles. If this is cut vertically across its fibers, the difficulty of maintaining it in position is very great, and when an unusual strain such as coughing and vomiting, it probably is impossible to maintain it in position.

On the other hand, if this structure is cut transversely in the direction of its fibers, it may be split across the linea below, and by retracting the rectus muscle out of its sheath on either side, sufficient room may be acquired for

almost all operations upon the stomach or gallbladder, *etc.* When the wound is closed, its edges automatically come together. No tension is required to coapt them, and thus no strangulation of the tissues can occur.

This same principle may be employed in an incision we are using lateral to the rectus muscle, in which the rectus is retracted medially. This incision is employed on the right side for operations upon the biliary passages, and on the left side for operations upon the splenic flexure of the colon, and particularly for splenectomy. It begins near the midline, three to four inches above the umbilicus, and extends obliquely downwards and outwards, just below the rib margin, almost to the iliac crest, just posterior to the anterior superior spine. This is in the direction of the fibers of the internal oblique muscle. The anterior sheath of the rectus muscle is cut transversely and the muscle freed from the sheath for a short distance above and below. The incision is continued across the fascia of the external oblique for two to three inches in line with the skin incision, and the external oblique is retracted further, laterally. The rectus muscle is retracted toward the midline, and its posterior sheath is split from the linea alba, laterally, into the internal oblique and transversus muscles. The internal oblique is split and the transversus and peritoneum cut in the same incision. This gives a very advantageous exposure of the gallbladder, bile ducts, appendix and pyloric end of the stomach on the right side. The wound comes together without tension and is quickly and easily sutured in layers.

The incision may be used to advantage on the left side in operations for removal of the spleen, and affords a better exposure than the usual incisions and with no danger of disruption or weakening of the abdominal wall.

Our experience, as indicated here by a review of 710 consecutive upper abdominal incisions, is that, of the vertical incisions, 284 had nine disruptions, or 3.2 per cent; 15 herniae occurred, or 5.3 per cent. Of 426 transverse incisions (if I may call these transverse), we had no disruptions, and only one hernia was found, which occurred in a patient who had had an omentopexy performed for cirrhosis of the liver.

The suture material in these cases was not investigated. We know that disruptions occurred in the first series of vertical incisions in which silk was employed in conjunction with plain and chromic catgut. The suture material which has been used in these 426 cases has been either plain No. 1 catgut or No. 0 chromic catgut.

The time required for making the incision is longer but the patient is more comfortable following the operation, and the sense of security in the mind of the surgeon makes the extra effort well worth while.

REFERENCE

- ¹ Sloan, G. A.: A New Upper Abdominal Incision. *Surg. Gynec. and Obstet.*, **45**, 678, 1927.

DR. HOWARD LILIENTHAL (New York City, N. Y.): Speaking only of perfectly clean wounds, it must be obvious that an infection in a perfectly clean wound is usually carried in by the scalpel.

I do not use a scalpel. I use the diathermic knife, the electrical scalpel. It has to be used with care, speed and precision, and thus obviates infection from the skin, even the deeper layers.

DR. ROSCOE R. GRAHAM (Toronto, Canada): An analysis of our wound infections and wound disruptions has shown a higher incidence than in the series which is being reported by Doctor Whipple. While Doctor Whipple is placing great emphasis on the type of suture material, and making a plea for

the efficacy of silk in wound closures, the actual type of material is probably not as important a factor as he would lead us to believe. One has but to see Doctor Whipple operate, to recognize the gentleness and care with which he treats tissues, and this I think, in no small way, has contributed to the excellence of his results. In our own series we have had no incidence of wound infection in which there was not evidence of an accumulation of serum or imperfect hemostasis in the wound. The former we believe is due to massive ligatures or traumatic methods in opening the abdominal wall. In other words, meticulous, sharp dissection, having regard for anatomic structures and planes, the avoidance of mass ligatures, and the securing of perfect hemostasis are probably the important factors in securing firm, primary healing of abdominal wounds. These above requirements must of necessity be fulfilled if the surgeon employs silk, and in this regard, the use of silk making necessary such a type of technic constitutes its greatest virtue.

DR. WALTER D. WISE (Baltimore, Md.): I would like to call your attention to a subject that I hesitate to bring before this Association, except to pass it through this organization to many of the younger surgeons. It is about the matter of knots.

One constantly sees, if one is alert to that subject as some of us have been taught to be by avocations instead of vocations, particularly that of sailing, the indifference with which surgeons tie knots. Speed, it seems, is what they are endeavoring to accomplish rather than meticulous care in tying a flat or reef knot.

This does not apply, of course, so much to silk as it does to catgut. In the use of silk, a granny knot will hold quite well, but it is not entirely reliable. In the use of catgut, anyone who has watched it swell and untie itself will realize that it is essential, not only to tie a reef knot, but to use a third throw.

What I am saying does not apply only to closing incisions, but it applies more particularly to the ligation of vessels, and probably accounts for some of the catastrophes.

It is probable that a good many disruptions of wounds, as has been hinted at but not actually said this morning, occur in the first day, or probably during the first hour or two after an operation, resulting from the act of vomiting or straining. That is the time, regardless of when the catgut digests or if there is any allergy or any other factors involved, when one wants the knot to hold. If the knots are tied with the indifference that one sometimes sees, then this may account for some instances of disruption.

DR. JOHN J. MORTON (Rochester, N. Y.): I would like to endorse Doctor Whipple's discussion on the healing of wounds because I have been using practically an identical technic during the last five years. I think that it should be emphasized that he does not make use of so-called retention sutures. I have given up using retention sutures also. The use of very fine silk, C grade, provides for accurate approximation of the divided tissues. There is one difference, however, in our technic. When we make a vertical incision, we go to the edge of the rectus muscle and retract the rectus over, so that we have our incision staggered and bolstered by the rectus muscle in front. I think this may help in some cases when postoperative distention occurs.

I have been very much pleased with this type of closure and I use it in gastric and gallbladder surgery as a routine. It is used on a good many other lower abdominal cases and even in some large bowel resections, when I am reasonably sure that there has been no major contamination.

DR. HARVEY B. STONE (Baltimore, Md.): There is one factor which has been mentioned only passingly, and which I think needs at least a word of

reference: that is, those cases of wound disruption which occur, apparently due to a failure of the healing power of the tissues, and which I believe in some instances have no relation either to the type of incision made or the material employed in the suture, or any of the other defects which have been mentioned previously.

I am confident that there are wounds which for some unknown biologic reason do not heal and I think in those cases, no mere technical procedure is going to correct that failure.

In the past, it has seemed to me that the incidence of such wounds has been strikingly high in patients suffering with advanced malignant disease, and in old age, conditions in which the recuperative power of the tissues is naturally lowered. These disruptions often take place in wounds 10 to 12 days after operation, when everything seems to have been progressing normally. The wound suddenly splits open during an attack of coughing or sneezing; when one examines the wound edges, they appear as though made only a day or two before, without any evidence of effective granulation.

Since the routine employment of blood transfusion in operations for malignant disease, it seems to me that the incidence of such disruptions has been remarkably decreased. One might infer that there was a factor that might be detectable in such cases, such as a diminished blood protein as suggested by Doctor Whipple, or some other dyscrasia, that accounts for this failure to heal. Only yesterday, I saw a patient who had had a small fibroma of the skin removed two weeks previously from the thigh; a small wound, two or three inches long, closed with silk. It had apparently healed and the dressings had all been removed. On the thirteenth day, the patient, while driving an automobile, simply cracked the wound wide open, throughout its entire extent. It didn't bleed much, and the cut surfaces looked almost as fresh as though it had been made just the day before.

DR. HUGH H. TROUT (Roanoke, Va.): There are a great many objections to the use of catgut, one of which has not received the attention it deserves. For example, for years we have been thinking that the degree and rate of absorbability of catgut were dependent largely upon chromic or tannic acid. The factor to which I refer is the age of the animal from which the gut is removed.

Our attention was first called to this by the report of Bulloch on Suture Material, made to the Royal Society of Surgeons of England. Since this time we have been doing considerable experimental work, trying to obtain a substitute for catgut, as well as test the absorbability of the various brands and sizes of catgut. Naturally, we have found that the older the animal the less absorbability the gut has. In fact, if one takes an old ram, the gut removed is practically a foreign body. Apparently it is impossible for the manufacturers to know definitely the age of the animals from which the gut is removed.

DR. CHARLES C. LUND (Boston, Mass.): Doctor Stone's remarks have stimulated me to enter this discussion. The work that I am going to mention is so incomplete that I was not planning to say anything about it at this meeting.

As many of you know, Wolbach and others, in 1926, demonstrated that wounds in animals with scurvy will not heal. Recently, determinations of blood vitamin C, the active principle in orange juice, have become reasonably easy to make and are reasonably accurate. At Doctor Cutler's Clinic and in London, it has been shown that the great majority of patients that were being

treated for gastric ulcer have a very low blood vitamin C. Some of them practically have scurvy.

At the Boston City Hospital, on our service we have now made something over 1,000 determinations on several hundred patients with miscellaneous surgical conditions. We cannot report any results as yet, except to say that in the population there are a great number of people of all classes who are running suboptimum levels of vitamin C, many of the levels apparently close to scurvy. Of course the level of serum protein, as mentioned by Doctor Stone, is also important in wound healing.

I think that vitamin C is probably also very important. I will say, however, that the first disruption that occurred in a patient who had a blood vitamin C determination made had an absolutely normal level. We checked up with the house officers and we found that, according to the requirements set forth by Doctor Whipple, this wound had been very, very badly sutured.

DR. PHILEMON E. TRUESDALE (Fall River, Mass.): Doctor Wise has called our attention to the undesirability of knots in wounds. There is a distinct disadvantage in leaving a field of operation studded with knots, especially when the material is chromicized catgut. The knot acts as a foreign body. After taking cultures from the wound before secondary closure, it will invariably be found that the wound is infected, if at all, around the knot. Bacteria may be found in the region of the knot and nowhere else.

There is always some degree of round cell infiltration at the point of fastening. The coarser the ligature, the greater the reaction. On the contrary, a knot which has been tied with fine silk or plain catgut produces very little reaction. A knot tied with No. 1 or No. 0 chromic catgut causes only a mild reaction, with a few bacteria, but when a No. 3 or No. 4 chromic catgut is used, there follows a marked reaction around the ligature. That is why at either end of the wound one frequently feels a hard, tender swelling, due to inflammatory reaction around the ligature.

DR. ALLEN O. WHIPPLE (closing): Regarding the results that Doctor Singleton has obtained by the incision that he uses: We have employed this incision in a number of upper abdominal cases and for gastric work, particularly around the pylorus, and we have found it an exceedingly good one.

In regard to the use of the electric cutting current: We have employed this at times, but it always seemed to me that there was more of a margin of tissue necrosis, even though the current is used quickly, as Doctor Lilienthal has suggested. We still prefer the scalpel incision.

Relative to knots, I am sure that all that has been said is correct about the tying of knots. Certainly in fine suture material, whether it be silk or catgut, a knot can be tied more securely and with less foreign body reaction than when the heavier grades are employed.

I did speak about the low serum protein in the cachectic individual with very poor wound healing. I am sorry that I did not bring that observation out more fully. Many of the points, because of the time, I was not able to deal with as fully as I would have liked to.

Doctor Harvey and Doctor Morton have really touched upon the important feature of wound healing, and that is: That in order to get optimum wound healing, whether one uses silk or catgut, one must minimize the tissue damage, and if one uses silk, the philosophy that goes with it develops inevitably and tends to minimize constantly tissue damage.

SPONTANEOUS RUPTURE OF THE SUPERIOR AND INFERIOR EPIGASTRIC ARTERIES WITHIN THE RECTUS ABDOMINIS SHEATH

ROBERT L. PAYNE, M.D.

NORFOLK, VA.

SPONTANEOUS rupture of the internal mammary or epigastric vessels within the rectus muscle is a definite surgical entity, though rarely occurring, and hence lacking the familiarity characteristic of the average abdominal lesion. Examples of this condition are appended:

CASE REPORTS

Case 1.—Male, age 55, had been driving an automobile over smooth roads for about four hours, after which time he turned the wheel over to his wife and settled back in the seat to rest his eyes and take a nap. Without any accountable reason, he suddenly became conscious of a mild, but sharply acute pain in the left side of his abdomen. This pain, at the end of 30 minutes, had increased to one of marked severity and the patient, being a doctor, feared that he had suffered an intestinal rupture or possibly a mesenteric thrombosis. There was no great shock, but he became nauseated and vomited. Two hours elapsed from the onset of the symptoms before he reached his home and received medical attention. The attending physician made a tentative diagnosis of some acute intra-abdominal lesion. He was taken to the hospital immediately, and was seen by the author shortly after admission.

Physical Examination disclosed a heavy-set, stout man, suffering with very acute abdominal pain, so severe that he could not tolerate the weight of the bed clothes on his abdominal wall. There was a mass in the region of the left rectus muscle at about the level of the umbilicus which was exquisitely tender on palpation, and appeared to be about the size of a grapefruit. The left rectus muscle was tensely rigid above and below the mass, but the rigidity seemed to stop with a clear-cut line of demarcation along the midline of the abdomen, and the right rectus and right half of the abdomen were neither tender nor rigid. The left side of the abdomen showed some tenderness and rigidity spreading out into the oblique and transversalis group of muscles, but these phenomena tapered off completely before reaching the anterior superior spine of the ilium. Temperature, 100.6° F.; pulse, 120; blood pressure, 175/95. R.B.C., 4,500,000, hemoglobin, 90 per cent; W.B.C., 14,000, polys., 82 per cent. The bleeding and coagulation time were normal, and there was no reduction from the normal in the platelets.

The abdomen was not distended. Nausea and vomiting had ceased; there was no mass to be felt by rectum, and the urine was negative for red blood cells and pus. Careful consideration was given to the possible diagnosis of incarcerated hernia, volvulus, mesenteric thrombosis or a ruptured viscus. Roentgenologic examination, made in both the supine and prone positions, revealed no evidence of subcutaneous emphysema or free air in the peritoneal cavity. *Diagnosis:* Spontaneous hemorrhage in the left rectus muscle.

Operation.—An incision six inches long was made through the skin overlying the left rectus muscle, with the center of the incision at about the level of the umbilicus. On reaching the anterior sheath of the rectus muscle, there was immediately observed an extensive ecchymosis with dark discoloration such as one would see underneath the peritoneum overlying a ruptured, ectopic pregnancy. Incision of the anterior sheath of the rectus disclosed a large, blood-filled cavity in which one could easily pass the entire hand both upward and downward. A quart of clotted blood was removed, and several bleeding vessels were found and ligated. Two of these bleeding vessels spurted,

one from above and one from below, and were thought to represent the anastomosis between the internal mammary and epigastric arteries. The rectus muscle appeared badly mutilated as a result of this rapidly dissecting hematoma. Much of the muscle had already undergone separation and pressure necrosis. The wound was closed except for one small Penrose drain for 48 hours. Recovery was uneventful.

Case 2.—White, male, age 64, was admitted to the hospital with the history of having had a slight pain in the left lower abdomen during the previous week. On the day of admission the pain had become suddenly and acutely severe, and had been continuous since the exacerbation began. The sudden development of pain occurred while the patient was at the breakfast table. He had taken no undue exercise since arising from bed, and there was no history of direct or indirect trauma. He had been nauseated for several hours before admission, but there had been no vomiting.

Physical Examination.—The abdomen was flat and scaphoid except over the left rectus muscle which stood out prominently from the symphysis almost to the ensiform, like a large Bologna sausage. The area of distention seemed to be absolutely limited by the external and internal boundaries of the sheath of the rectus muscle. Rigidity was board-like, whereas, the oblique muscles to the outer side and the right rectus and right half of the abdomen were soft and flaccid. There was a history of the patient having stuck a nail in the plantar surface of the left foot eight days previously. Temperature, normal; pulse, 72. R.B.C., 4,100,000; W.B.C., 10,600, polys., 80 per cent. There was no increase in any of the reflexes, and no spasm or stiffness of the jaws or neck. The normal temperature, moderate leukocyte count, and the slow pulse were not in keeping with an acute intra-abdominal condition. Because of the history of a puncture wound of the foot eight days previously, it was thought that this might possibly be a case of localized tetanus. The wound in the foot appeared to be completely healed, and showed no tenderness or inflammation; it was, however, opened by blunt dissection, but no evidence of inflammation, serum or pus was found. Cultures were taken from the wound and tetanus antitoxin, 1,500 units, injected around the wound; 20,000 additional units were given in the vein, and 20,000 in the muscle. During the next four days 280,000 more units of tetanus antitoxin were administered, during which interval of four days there had been no change in the patient's condition. There had been no further evidence of tetanus, and the culture from the wound in the foot was negative at the end of five days.

The left rectus muscle had, however, become more tense, and more sensitive, necessitating the administration of sedatives and opiates to control the pain. There then appeared a slight, globular swelling at about the center of the left rectus sheath, aspiration of which disclosed old, dark blood which proved negative on culture. In the meantime, the patient had developed a rather septic type of temperature which ranged from normal in the morning to 103° F. in the evening. The bleeding and coagulation time were normal; platelets, 275,000; leukocytes, 7,100, polys., 72 per cent. Blood pressure, 148/88. It was evident that the patient had been upset by the large doses of tetanus antitoxin administered because of an erroneous diagnosis.

Operation.—An incision five inches long was made over the center of the left rectus muscle. The sheath was almost black from hemorrhagic extravasation; upon incision of it there was evacuated a large quantity of old, broken down and liquefied blood clots. The entire rectus muscle adjacent to the incision seemed to be completely destroyed by pressure necrosis, and by dissection of the hemorrhage upward and downward between the muscle fibers. There was only one bleeding vein seen, and this followed the enucleation of some of the organized, old adherent clot. Cultures from the cavity in the rectus muscle showed colon bacilli and unidentified Cocci after three days. The patient was immediately relieved of his pain. The temperature became normal, and convalescence was rapid and uneventful.

Case 3.—Female, age 46, was referred to the author by her family physician, who thought she had a pelvic tumor on the left side which was undergoing degeneration because of a mass, the presence of acute tenderness, and some little fever. The patient was

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a multipara whose menses had been perfectly regular, and there was no pelvic dysfunction or symptoms of pelvic disturbance in her history. She related that this trouble began with pain in her lower left abdomen one week previously, and that it had steadily progressed until she appreciated that there was a tender mass in her lower abdomen on the left side.

Physical Examination of the pelvis showed a normal cervix by inspection, and bimanual manipulation disclosed the uterus to be freely movable; no tenderness in the cul-de-sac, or in her broad ligaments. The pelvis to the right of the midline was soft on pressure, but a large mass was palpable to the left of the midline, and was very tender; this tumor could only be indefinitely appreciated by the fingers in the vagina.

On abdominal examination, there was at once observed a crescentic area of purple ecchymosis extending below the umbilicus for about two inches, and which was confined entirely to the lower half of an imaginary circle drawn around the umbilicus. The patient was questioned carefully as to how she had gotten this bruised area in the skin. Any history of injury or any other predisposing cause was denied by the patient. Furthermore, she did not know that this area of discoloration existed. Further examination disclosed a mass about the size of an orange lying apparently within the rectus sheath between the level of the umbilicus and the pubic bone. The mass was movable, and quite tender, while the muscles to the right and left of the mass were perfectly soft and not painful to pressure. Upon asking the patient to assume a half sitting posture, it immediately became apparent that the mass became more prominent and was apparently located in the anterior abdominal wall. The ecchymosis around the umbilicus was the real clue to the diagnosis of hematoma in the rectus muscle. It was assumed that some of the blood from the hematoma had dissected down below the semilunar fold of Douglas, and then had gravitated along the obliterated hypogastric artery upward to the umbilicus. Operation disclosed an hematoma in the lower rectus sheath; this had become organized, and when enucleated was about the size of a small orange. The patient was immediately relieved of her pain and made an uneventful recovery. Before leaving the hospital, and after a most careful questioning, she finally thought that perhaps the beginning of this trouble was a sneezing spell. However, she was not positive in her statements, and it did seem that this case should be considered a spontaneous rupture of the vessels.

Symptomatology.—In spontaneous rupture of the epigastric artery, the patient usually complains of a sudden, severe pain to the right or left of the midline, and usually at about the level of the umbilicus. However, premonitory soreness of mild character lasting from six to seven days has often been described preceding extensive vascular rupture with massive hemorrhage. This type of onset has been observed in several cases, notably those reported by McCarty,¹ Culbertson,² and in one of the author's cases. With the rapid development of massive hemorrhage the pain is usually very severe, and the patient will not tolerate the weight of clothing or any extensive manipulation in the examination. There is usually a normal or slightly increased temperature, and a moderate leukocytosis. There is usually some prostration, and frequently prolonged nausea and sometimes vomiting. There is usually a localized mass, exquisitely tender to palpation, and described in many reports as varying in size from a hen's egg to that of a large grapefruit. The mass is always confined to the sheath of the rectus muscle, but it must be remembered that there is no posterior sheath below the semilunar fold of Douglas, and hence blood may extravasate downward anterior to the peritoneum.

Several cases reported have shown the hematoma to have pushed the anterior peritoneum inward until the mass could be palpated per vaginam.

Ecchymosis is a most important sign, and frequently offers the first intimation as to the correct diagnosis (Case 3). Vernon³ reported a case with suggillation about the pubis and perineum. One of the characteristic features about the mass is that it does not change its position, and always appears fixed in the right or left abdominal wall.

Fothergill⁴ contributes a sign which was demonstrated in Case 3. If the recti muscles are made to contract by having the patient sit partially up, the mass can still be felt and yet it cannot be moved to either side of the abdominal wall. The absence of rigidity and tenderness in the abdominal wall adjacent to the tumor is almost pathognomonic of a lesion in the rectus sheath, in contradistinction to what would be expected if there were an intra-abdominal lesion present. Tenderness, tonic contraction on one side, and absence of surrounding rigidity are definitely characteristic of this lesion.

The differential diagnosis is considered very important by most authors writing on this condition. They seem to feel that the lesion has been too frequently diagnosed as an acute intra-abdominal lesion. It is true that in many of the reported cases the preoperative diagnosis has been mesenteric thrombosis, intussusception, volvulus, incarcerated hernia, gallbladder disease, twisted ovarian cyst, ectopic gestation, and degenerating fibroids of the uterus. McCarty feels that it is particularly important to recognize the possibility of this condition being associated with pregnancy, lest there be confusion with an ovarian cyst or a pedunculated fibroid, as cases have been reported occurring during pregnancy, labor and in the puerperium.

Etiologic Factors.—This covers a rather broad field of assumption. Many cases, however, are reported to have had as a contributing factor, coughing, sneezing, and any severe jolting associated with sudden muscular contraction. Some cases have been reported in association with infectious processes such as tetanus, tuberculosis, typhoid and typhus fever, influenza and low states of muscular inanition, such as is found in chronic ulcerative colitis. It seems quite reasonable to expect that a weak, atrophic muscle would require less effort to produce either a tear of its fibers or rupture of the blood vessels. Conversely, one might intelligently inquire whether atrophic muscles are capable of contracting sufficiently to produce rupture of the normally elastic vessels.

Degeneration of blood vessels must also be considered an important contributing factor, and it is to be remarked that in practically all of the cases reported, the individuals are usually in late middle life, and all showed evidence of sclerosis and vascular degeneration as indicated by varying degrees of hypertension. In 50 case reports, personally reviewed, the average blood pressures were 170/95. One would expect muscular atrophy and vascular degeneration to be present in association with some infectious processes, particularly in men leading a sedentary life, and in inactive women, especially during pregnancy and the puerperium. As indicative of how little may be

the contributing factor, Halperin⁵ reports the occurrence in a woman, age 71, resulting from raising up as she turned over in bed. As a rule, however, no definite cause can be found for the so-called spontaneous case. Malpas⁶ thinks that the spontaneous ruptures are due to some latent blood dyscrasia. He reports two such suggestive cases. Evidence of this condition was not present in my patients, and was noted in only three of the other cases reported in the literature. Many authors think the contributing factor is a pendulous abdomen, which produces traction upon the vessels and thus induces chronic attenuation and friability. A considerable effort has been made by many surgeons to study the histologic pathology of the muscle and vessels found at the time of operation; the most characteristic findings have been old hemorrhage, lymphocytic infiltration and necrosis. On the other hand, Trofimoff¹⁴ and numerous others could find no satisfactory explanation in the excised muscles and vessels involved in the hematoma. Nørgaard¹⁵ studied 72 cases in which the correct diagnosis was made in only 11 instances. The only contributing cause he could determine was a low capillary resistance and in two cases a prolonged bleeding time. Giardina¹⁶ records one case in a syphilitic who had an associated pyloric ulcer, the microscopic studies from the hematic area in the rectus showing a chronic productive myositis. One of our cases showed a localized arteritis and peri-arteritis (Case 1), and a number of case reports have recorded arteriosclerosis or hypoplasia of the vessels, including focal degeneration of muscle and vessels following infections. Two reports showed aneurysmal dilatation, calcification and atheroma in the inferior epigastric arteries.

Quite an important group are those occurring in pregnancy, during parturition, or soon after delivery. Spirito¹⁷ and others have reported the sudden development of a hematoma in the rectus muscle occurring 15 to 30 minutes after the expulsion of the placenta, while Maxwell¹⁸ reports 12 spontaneous cases occurring during pregnancy, and 46 cases from other causes, in none of which was correct diagnosis made before operation. The frequent occurrence in multipara is in keeping with the theory of muscular stretching and impairment as a causative factor, the acute lesion being ushered in with an episode of coughing or sneezing. Jaschte and Meldolesi¹⁹ note arteriosclerotic changes in the epigastric vessels, especially after repeated pregnancies. To controvert this, Dencks²⁰ observed an instance which fell in this category in a woman, age 65, and attempted to determine the pathology of such cases by examining the histology of the epigastric arteries and rectus muscles in 95 cadavers of both sexes ranging in age from 50 to 90. The findings were not outstanding: Some arteriosclerotic changes were determined, but were not of such localized high grade character as to indicate enough friability to be conducive to spontaneous rupture.

Infection has played an important part in the study of the localized pathology. Chaliar and Vallery²¹ reported an infected hematoma of the rectus muscle following typhoid fever in which the cultures showed Eberth's bacillus. Kenwell²² reported an infected hematoma in which the cultures demonstrated

Staphylococci aureus hemolyticus. Wehik²³ reported 20 cases that he had collected of infected hematomata, but does not mention the infecting organisms. Harris,²⁴ operating under a mistaken diagnosis of acute appendicitis, found an infected hematoma of the right rectus muscle, and cultures disclosed a gram-positive *Bacillus* which was not identified. Delay in diagnosis and in operative treatment results in liquefaction of the hematoma with secondary infection of the clots by *Staphylococci* or the colon groups as demonstrated in Case 2. Influenza is the most outstanding infection found associated in the various case reports reviewed, hematomata having developed in the rectus muscle as a complication.

With²⁵ drew a most interesting conclusion in a case studied and operated upon by him. He thought the underlying factor was a hemorrhagic diathesis due to C avitaminosis. Study of the blood in this case showed a total absence of ascorbic acid. No estimation of vitamin C in the urine was made. Several authors have thought that systemic disease had an important bearing on the development of the hematoma. Lehman²⁶ reports a case associated with splenomyelogenous leukemia, and Dlugi²⁷ reports the development of a hematoma in the left rectus muscle, which produced intestinal obstruction in a case known to have leukemia. Del Carril²⁸ also records a case of intestinal obstruction, occurring in a three months old child, due to extraneous pressure from a large hematoma in the right rectus muscle. Lenner²⁹ records a most interesting case, occurring after a Pfannenstiel incision, and says: "There have been other reports of hemorrhage into the rectus sheath after Pfannenstiel's incision." It seems sound to conclude that hematoma of the rectus develops as a result of three causes: (1) Muscular effort; (2) following low-grade infection; and (3) spontaneous rupture due to focal degeneration of muscle and vessels.

The military surgeons of France, Germany, Austria and Russia have all recorded numerous cases following sudden muscular effort connected with military training. They conclude that rents of small vessels generally produce slowly growing tumors and a gradual exacerbation of symptoms. Rents of a large vessel produce a rapid development of symptoms and the pathognomonic signs are acute and definite. In this connection, Brendeau³⁰ reports a rapidly developing hematoma in the rectus muscle which subsequently ruptured into the peritoneal cavity, with immediate disappearance of the tumor. The rapidly developing tumors are always accompanied by signs of peritoneal irritation such as belching, nausea, distention, rigidity, and sometimes vomiting. In the slowly developing case the mistake is usually made of diagnosing an intra-abdominal tumor, and if the mass is intramural, it is usually confused with an incarcerated hernia or tumors of the abdominal wall produced by sarcoma, fibroma, desmoid, gumma, tuberculosis or actinomycosis. The most suggestive finding in the slowly developing case is Laffont's sign, namely, discoloration around the umbilicus which has been referred to previously, and was present in Case 3.

Pathology.—There was no informative record covering the underlying pathology of this condition. It is significant that Beals, Blanton and Eisen-drath⁷ found eight cases among 140 bronchopneumonias which came to autopsy. They thought the contributing factor was some localization of the infection in the rectus muscle associated with beginning abscess formation followed by rupture and hemorrhage incident to violent coughing. In the pathologic report of Behan's⁸ case there was shown to be an alteration in the muscle fibers, consisting of an exaggerated granulation and hyalin degeneration, suggesting a degenerative process prior to the rupture. The recent hematoma appeared to have been extravasated between these degenerated fibers. The pathologic diagnosis was "chronic myositis." One might ask if the pathologic reaction in this case was contributory to rupture and hemorrhage, or conversely, were the muscle changes above described due to pressure necrosis from the hematoma.

Vascular disturbance followed by hyalin degeneration is the principal pathologic lesion associated with rupture of muscle or vessels within the rectus sheath. This condition seems to occur more frequently within the rectus muscle than in any other single large muscle of the body. Brödel,⁹ in a most scientific treatise on the anatomy of the rectus muscle, covers this problem in great detail. He shows that there are no main arterial trunks in the center of the rectus muscle, but on the other hand, a diffuse capillary bed through which active flow of blood is brought about by muscular contraction. This normal mechanism is often disturbed by pathologic blocking of the capillary beds with resulting hyalin degeneration. This is thought to occur frequently in mild, acute infections, or in healthy individuals without ever being detected. We know that injured or degenerated muscle fibers promptly regenerate, and in a short period of time nothing can be demonstrated either clinically or microscopically. However, regeneration does not take place if there has been a massive injury resulting in a large area of hyalin degeneration of the muscle. Brödel thinks that this condition in a large area, or in numerous small areas scattered throughout the muscle, is conducive to rupture of the muscle fibers or the vessels. The more resistant arteries are less liable to rupture than the frail, thin-walled veins. Any interest whatever on this subject should certainly stimulate one to read Brödel's article.

Anatomy.—The principal function of the rectus muscle is regulating the intra-abdominal pressure and aiding the other associated muscles in the act of expiration. It is said that voluntary muscles are capable of contracting down to one-half their length. Brödel quotes the famous artist, Leonardo da Vinci (1452-1519), as stating that the rectus muscle during flexion and extension can stretch and contract as much as nine fingerswidth, which corresponds in the rectus muscle of an athletic youth to about 17 cm. This contraction is controlled by the three transverse tendons which really divide the rectus muscle into four segments, thus providing for segmental contractions in contradistinction to one large central contraction.

The nerve supply comes from the anterior branches of the fifth to the

twelfth thoracic nerve while the vascular system is composed of the terminal branches of the internal mammary commonly spoken of as the superior epigastric artery, while from below, the inferior epigastric ascends to anastomose with the terminal branches of the internal mammary by a very fine capillary plexus. In addition, the central portion of the muscle is further supplied by small arteries coming from the seventh, eighth and ninth intercostals which anastomose with the epigastric plexus. There is a double set of veins accompanying each arterial system; the whole vascular network is situated on the dorsal surface of the rectus muscle.

Incidence of Rupture.—The impression is almost universal that spontaneous rupture of the epigastric artery is a very rare occurrence. The following facts taken from the literature controvert this idea: Maydl,¹⁰ in 1882, reviewed the literature from 1809 to 1880, and stated that the condition was accurately described by Hippocrates and Galen. Reference is again made to Leonardo da Vinci's description previous to 1519. Maydl collected 14 cases of spontaneous rupture reported previous to 1880, and Cullen¹¹ is of the opinion that one of the first cases described in this country was by Richardson,¹² in 1857. Wohlgmuth¹³ collected 127 cases up to 1923, 79 of which were below the navel, and 18 below the semilunar fold of Douglas. A study of the literature appearing during the last decade affords 77 articles on this subject, and presents the records of 165 cases for the analysis of the histories, operative findings, and pathologic investigations. There are probably twice this many cases which occurred during this period that have been observed but which were not reported.

Treatment.—Early and correct diagnosis, followed by prompt operative evacuation, is the proper treatment. The seriousness of hematoma in the rectus muscle, whether developing spontaneously or otherwise, seems to have been greatly exaggerated, in my opinion, in the articles that have been reviewed. Among the 165 cases reported, there are only three deaths recorded: two from intestinal obstruction reported by Dlugi and del Carril, and one reported by MacLennan³¹ of a man, age 55, in whom the diagnosis of acute intestinal obstruction was made, and nothing found but a hematoma in the middle third of the left rectus muscle. Death occurred on the fourth day, and necropsy showed a hemorrhage to have again recurred; throughout the arterial system there was an advanced degree of arteriosclerosis. The principal danger in the treatment of these cases seems to lie in a mistaken diagnosis, followed by unnecessary procrastination relative to operation. In a large percentage of these delayed operations, infection of the hematoma is superimposed and thus becomes a grave complication. With regard to delayed diagnosis and treatment in these cases, Giese³² reports a most interesting case in which medicolegal aspects assume considerable importance. The patient was a woman, age 86, unhappily located in the home of a couple who were quite unkind to her. She lay abed for two days and died without any physician having been in attendance. Gossip was rife, and the village pastor urged autopsy. The most prominent physical finding was a band of dark

blue discoloration 8 cm. wide, on the left side, and extending from the umbilicus to the pubes. Incision yielded dark blood. External violence was charged. Giese was called in to make further examination and found extensive arteriosclerotic changes in various parts of the circulatory system, and also the presence of pneumonia. The verdict was that the extravasation of blood in the muscle occurred as the result of spontaneous seepage, and that death had been due to pneumonia. He does not explicitly mention rupture of the deep epigastric or internal mammary arteries. Considering the frequency of hematoma developing in the rectus muscle and the apparent lack of familiarity with this lesion, it seems pertinent to recall the admonition of Mr. Mailer,³³ who appropriately said that while the abdominal cavity is called Pandora's box, it is highly important that we do not forget "the lid."

CONCLUSIONS

So far, we can only speculate about the causes of hemorrhage into the sheath of the rectus muscle and point to certain predisposing factors. The primary etiologic possibilities may be found either in the muscle itself or in its vessels.

Muscle.—Degenerative changes in muscle fibers, predisposing to rupture, are described by several authors, but we should not forget that degenerative changes in muscle fibers can be the consequence of hemorrhage. As to the peculiar localization of this hemorrhage, the nearly unique position of the muscles of the abdominal wall, especially of the rectus, may be pointed out. Except for the small muscle group of the cheek, the muscles of the abdominal wall, and especially the recti, are the only muscles of the human body not supported by an underlying bone. We know that Zenker's degeneration localizes mainly in this muscle, and that this type of degeneration does not only occur in typhoid fever, but also as the result of other infectious conditions. In addition, we know that pregnancy, another of the predisposing factors, leads to both degeneration and regeneration of muscle fibers in the abdominal wall and into an occasional microscopic rupture of the fibers, and that these changes are more pronounced in cases which have had infection during pregnancy. This was proved by Strauss³⁵ in 20 postmortem examinations made during the last month of pregnancy. The peculiar situation of the rectus muscle, infection and pregnancy, may have to be counted among reasons for primary change in the muscle, but to me the other factor—vascular change—seems more important, although any one of the components may play its part.

Vessels.—Hemorrhage may arise either from arteries, veins, or from capillaries, and be precipitated by rupture or by diapedesis. When we consider the causes of vascular changes which may have certain connections with the occurrence of this disease, the following may be considered:

(1) *Arteries.*—Arteriosclerosis; syphilis; necrosis of the media, the so-called "third disease" of the aorta which leads to spontaneous rupture of this vessel.

- (2) *Arteries and Veins*.—Infectious changes.
- (3) *Veins*.—Changes in pregnancy.
- (4) *Capillaries*.—Spasm of the arteries leading to anemia, with consecutive damage of the capillaries, which results after release of the spasm, in hemorrhage (diapedesis) from capillaries—one of the mechanisms considered in cerebral apoplexy.

Arteriosclerosis, not unlikely, plays its part because the disease in question is likewise one of old age. Syphilis has not been found in these cases. Necrosis of the media is only mentioned incidentally, as we do not know of similar changes occurring in arteries other than the aorta. Infectious changes may play a greater part. They were often present in persons with hemorrhage into the rectus muscle, and in many cases, especially, it was influenza which, according to Stoerk and Eppstein,³⁶ frequently led to vascular changes. In support of the capillary mechanism, we may point to the frequently found ecchymosis; often, however, these may not be differentiated from suggillation, while on the other hand, hypertension is not among the concomitant diseases. Changes in veins, predisposing to hemorrhage, are not uncommon in pregnancy. Varices develop in the abdominal wall as well as in the legs, where only mechanical causes need be considered.

Today, therefore, we are, apparently, not able to determine the definite cause of hemorrhage into the rectus muscle. The possibilities mentioned above may be helpful in further study. We can point to predisposing factors such as old age, pregnancy and infection, and the not infrequently found combination of the latter with one of the former.

BIBLIOGRAPHY

- ¹ McCarty, R. B.: *Am. J. Surg.*, **23**, 480-483, March, 1934.
- ² Culbertson, C.: *J.A.M.A.*, **85**, 1955, December, 1925.
- ³ Vernon, S.: *J.A.M.A.*, **98**, 2199, June 18, 1932.
- ⁴ Fothergill, W. E.: *Brit. Med. Jour.*, **1**, 1941, 1926.
- ⁵ Halperin, G.: *Surg., Gynec. and Obstet.*, **47**, 861-863, December, 1928.
- ⁶ Malpas, P.: *Brit. Med. Jour.*, **1**, 1130-1131, June 21, 1930.
- ⁷ Beals, Blanton and Eisendrath: *J.A.M.A.*, **72**, 850, 1919.
- ⁸ Behan, R. J.: *Bost. Med. and Surg. Jour.*, **182**, June 24, 1920.
- ⁹ Brödel, M.: *Bull. Johns Hopkins Hosp.*, **61**, 295-312, November, 1937.
- ¹⁰ Maydl, C.: *Ztschr. f. Chir.*, **17**, 306-361; 513-547, 1882.
- ¹¹ Cullen, T. S.: *Bull. Johns Hopkins Hosp.*, **61**, 317-348, November, 1937.
- ¹² Richardson, S. B.: *Am. J. Med. Sci.*, **33**, 41, 1857.
- ¹³ Wohlgmuth, K.: *Arch. f. klin. Chir.*, **122**, 649, 1923.
- ¹⁴ Trofimoff, A. M.: *Vestnik khir.*, **19**, 286-294, 1930.
- ¹⁵ Nørgaard, F.: *Hospitalstid*, **80**, 246-251, March 2, 1937.
- ¹⁶ Giardina, S. G.: *Rassegna internaz. di clin. e terap.*, **10**, 677-686, August, 1929.
- ¹⁷ Spirito, F.: *Rassegna internaz. di clin. e terap.*, **13**, 787-805, August 15, 1932.
- ¹⁸ Maxwell, A. F.: *California and West. Med.*, **30**, 407-410, June, 1929.
- ¹⁹ Jaschte and Meldolesi: Cited by Bompiani in *Fisiologia e Patologia Clinica stato perale*, Toma, 1935, edit. Pozzi.
- ²⁰ Dencks, G.: *Deutsche Ztschr. f. Chir.*, **213**, 159-169, 1929.
- ²¹ Chaliel and Vallery: *Lyon Med.*, **140**, 493-495, November 6, 1927.
- ²² Kenwell, H. N.: *New York State J. Med.*, **29**, 1186-1189, October 1, 1929.

- ²³ Wehik, M.: *Acta chir. Scandinav.*, **53**, 531-544, 1928.
²⁴ Harris, R. I.: *Canadian Med. Assn. Jour.*, **14**, 1739, 1924.
²⁵ With, S.: *Hospitalstid*, **80**, 521-522, May 4, 1937.
²⁶ Lehman: Personal communication.
²⁷ Dlugi, H.: *Polska gaz. lek.*, **14**, 562-563, August 4, 1935.
²⁸ del Carril, M. J.: *Prensa med. argent.*, **23**, 1982-1984, August 19, 1936.
²⁹ Lenner, A.: *Acta obst. et gynec. Scandinav.*, **15**, 475-490, 1936.
³⁰ Brendeau, A.: *Gynec. et obst.*, **30**, 168-169, August, 1934.
³¹ MacLennan, D.: *Brit. Med. Jour.*, **1**, 895, May 26, 1928.
³² Giese, E.: *Deutsche. Ztschr. f. d. ges. gerichtl. Med.*, **23**, 304-308, 1934.
³³ Mailer, R.: *Brit. Med. Jour.*, **1**, 637, March 28, 1936.
³⁴ Walmsley, R.: *Jour. Anat.*, **71**, 404-414, April, 1937.
³⁵ Strauss, Arnold: *Virchow's Arch.*, **266**, 4, 1927.
³⁶ Stoerk and Eppstein: *Wien. klin. Wchnschr.*, **32**, 1086, 1919. *Frankfurter leitschr. f. patholog.*, **23**, 163, 1920.

DISCUSSION.—DR. MONT R. REID (Cincinnati, Ohio): I feel that I express the sentiments of this society when I express my personal appreciation to Doctor Payne for his excellent discussion of the subject.

The occurrence of this condition in a large charity hospital, such as ours, is frequent enough to bring the subject up for general discussion once every year or so. Yet, as so well expressed by Doctor Payne, the reason for the discussion is that the true condition is not thought of and the patient is operated upon under the assumption that there exists some acute intra-abdominal condition. In a certain sense, then, this condition is analogous to acute mesenteric lymphadenitis or hemorrhage from a graafian follicle, which are so frequently operated upon for acute appendicitis. The analogy rests largely upon the failure of the surgeon to be conscious of, or think of, the occurrence of these conditions. I am quite sure that Doctor Payne's paper will serve to make us conscious of the occurrence of spontaneous apoplexy of the epigastric and internal mammary arteries and, being conscious of this condition, to treat them with fewer instances of erroneous judgment. (I think I am quite safe in saying that a correct diagnosis will lead to an intelligent management of these cases by American surgeons and that a correct diagnosis depends largely upon a consciousness of the incidence of its occurrence.)

That we need look for no specific etiologic cause of this condition seems to me fairly obvious. The epigastric and internal mammary arteries are certainly not immune to those conditions of disease and trauma which make arteries subject to apoplexy in other parts of the body. Besides, the excellent anatomic studies of Cullen and Brödel show why these two vessels may be subjected to inordinate strain during the voluntary and involuntary functions of the body.

The medical profession has long regarded spontaneous arterial hemorrhages as being peculiar to the domain of the central nervous system. Doctor Payne has drawn attention to the fallacy of this assumption. Yet, I would add a word of warning, that they are not peculiar to the central nervous system and the rectus abdominis muscles; they may occur in any part of the body. On several occasions I have detected spontaneous leakage of arteries by hearing a course systolic bruit over the course of the peripheral vessels. I wonder if Doctor Payne has ever detected a bruit over hemorrhages into the rectus abdominis muscle?

Very shortly, my friend Dr. H. Glenn Bell, of the University of California, will report eight cases of spontaneous hematoma occurring in the rectus muscle. As so aptly expressed to me in a personal communication, he

wonders if minor occurrences of this condition are not frequently overlooked. Among people who normally live sedentary lives but exercise strenuously on Sundays and holidays, he has seen several who complained of severe upper abdominal pain, showed some rigidity of the rectus muscles and a few who showed the signs of an indefinite mass. Yet on most careful study no evidences of intra-abdominal abnormalities could be found. And the patients recovered completely. (It is often said that for every case of pancreatitis which is diagnosed, there must be hundreds which are not recognized; it may be that there are many cases of small unrecognized hemorrhages into the rectus abdominis muscles. Certainly the discussion of this whole problem makes me wonder about several cases which have puzzled me.)

THE REPLACEMENT OF SODIUM CHLORIDE IN SURGICAL PATIENTS*

FREDERICK A. COLLER, M.D., ROBERT M. BARTLETT, M.D., DERMID
L. C. BINGHAM†, F.R.C.S. (EDIN.), WALTER G. MADDOCK, M.D.,
AND SVEND PEDERSEN, PH.D.

ANN ARBOR, MICH.

FROM THE DEPARTMENT OF SURGERY OF THE UNIVERSITY OF MICHIGAN, ANN ARBOR, MICH.

THE IMPORTANCE of replacing the sodium chloride and water which may be lost from the body by such abnormal ways as vomiting, gastroduodenal suction, diarrhea, drainage from biliary and intestinal fistulae has been emphasized since the investigations of O'Shaughnessy,¹ Hartwell and Hoguet,² MacCallum, Lintz, Vermilye, Leggett, and Boas,³ Haden and Orr,^{4, 5, 6, 7, 8} Gamble and Ross,⁹ and many others,^{10, 11, 12} showed the value of such therapy. In practice, when a salt deficiency exists the amount of sodium chloride given has been largely empiric, one to five liters of saline solution being administered and the sodium chloride restoration followed by blood chemistry studies. This method has at least two faults. If the sodium chloride needs of the patient have been underestimated, valuable time is lost in restoring the body chemistry to normal; and secondly, if an excessive amount of salt is given, the error will not be shown by the blood chemistry studies and the patient may develop edema.^{13, 14, 15, 16, 17, 18} The purpose of this paper is to present briefly‡ data leading to simple accurate rules for: (1) The maintenance of a normal sodium chloride concentration in patients losing sodium chloride while under observation; and (2) the restoration of sodium chloride in patients whose sodium chloride concentration is below normal when first examined.

The Maintenance of a Normal Sodium Chloride§ Concentration.—In actual surgical practice the problem of maintaining a normal sodium chloride level deals mainly with the patient who, while in the hospital, is losing important amounts of water and sodium chloride through loss of gastro-intestinal secretions. In 1937, Dick, Maddock and Coller¹⁹ pointed out that the concentration of sodium chloride in these secretions is almost always less than the concentration of sodium chloride in physiologic saline or Ringer's solution, and they suggested that if one replaced the secretion loss by an equal volume of these solutions, a satisfactory water and salt balance should be maintained.

* This study was aided by a grant from the Rackham Research Fund for Graduate Studies, and the James and Elizabeth Inglis Fund for Surgical Research.

† Fellow of the Medical Research Council of Great Britain, Clinical Tutor in Surgery, Royal Infirmary, Edinburgh.

‡ A more complete presentation of sodium chloride metabolism in surgical patients will be published subsequently.

§ Throughout the study only the chloride ion was measured and, as is customary, its value was expressed in terms of sodium chloride.

To determine the value of this volume-for-volume rule a series of patients who were losing alimentary tract secretions were studied in the following manner: Immediately after operation each patient was weighed on a special scale, a blood specimen was taken for the determination of the plasma chlorides, a Levine tube was inserted into the stomach and gastroduodenal suction was instituted. Nothing was given by mouth and the water requirements for the day were provided for by the intravenous administration of 5 per cent glucose in distilled water. On the following morning the patient was weighed, the 24-hour specimens of urine and alimentary tract drainage were measured and their chloride content determined, and blood was again taken for a plasma chloride determination. A volume of physiologic saline or Ringer's solution equal to the volume of drainage for the previous 24 hours was then given intravenously, and in addition sufficient 5 per cent glucose in distilled water to provide for the water needs of the body. For the several days that this procedure was carried out, no stools were passed. The salt losses through the skin were not determined, but in no case was there profuse sweating. The

TABLE I

REPLACEMENT OF UPPER GASTRO-INTESTINAL SECRETION LOSSES WITH EQUAL VOLUMES OF
PHYSIOLOGIC SALINE SOLUTION

1 Liter \approx 8.5 Gm. NaCl										
Secretions Lost						Salt Given				
Patient	24 Hours Ending	Body Weight Kg.	G.I. Tract Cc.	Bile T-Tube Cc.	Total		Phys. Saline Cc.	Salt Content Gm.	Plasma Chlorides Mg. NaCl/ 100 Cc.	NaCl in Urin Gm.
					Volume Cc.	NaCl Gm.				
E. K.	2-18	—	—	—	—	—	—	—	556	—
	2-19	49.36	320	400	720	2.94	0	0	528	2.45
	2-20	47.86	850	290	1,140	7.50	708	6.03	528	1.10
	2-21	48.26	0	290	290	1.75	1,165	9.90	540	1.06
	2-22	47.67	0	250	250	1.49	287	2.44	540	1.11
	2-23	47.44	0	250	250	1.01	478	4.06	564	1.26
			1,170	1,480	2,650	14.69	2,638	22.43		6.98
E. M.	3-9	58.01	—	—	—	—	—	—	582	—
	3-10	59.06	900	0	900	6.46	0	0	490	1.21
	3-11	57.25	840	0	840	4.94	865	7.35	492	1.65
	3-12	56.85	920	0	920	5.69	835	7.01	505	1.51
	3-13	56.30	1,020	0	1,020	5.30	922	7.84	490	0.70
	3-14	56.50	0	0	0	0	1,030	8.76	513	0.80
			3,680	0	3,680	22.39	3,652	30.96		5.87
M. M.	2-21	42.01	—	—	—	—	—	—	571	—
	2-22	42.62	220	0	220	0.56	990*	5.86	533	6.85
	2-23	41.32	350	0	350	2.23	243	2.07	540	1.42
	2-24	41.65	390	0	390	2.45	354	3.01	541	1.11
	2-25	40.26	0	0	0	0	385	3.27	545	1.53
			960	0	960	5.24	1,872	14.21		10.91
M. A.	4-5	54.46	—	—	—	—	—	—	568	—
	4-6	—	490	160	650	4.12	0	0	490	2.17
	4-7	53.26	780	240	1,020	6.40	669	5.69	500	3.12
	4-8	51.29	910	230	1,140	7.63	1,010	8.59	512	1.59
	4-9	50.87	0	215	215	1.08	1,150	9.78	535	0.98
	4-10	50.92	0	220	220	1.14	393	3.34	545	0.76
			2,180	1,065	3,245	20.37	3,222	27.40		8.62

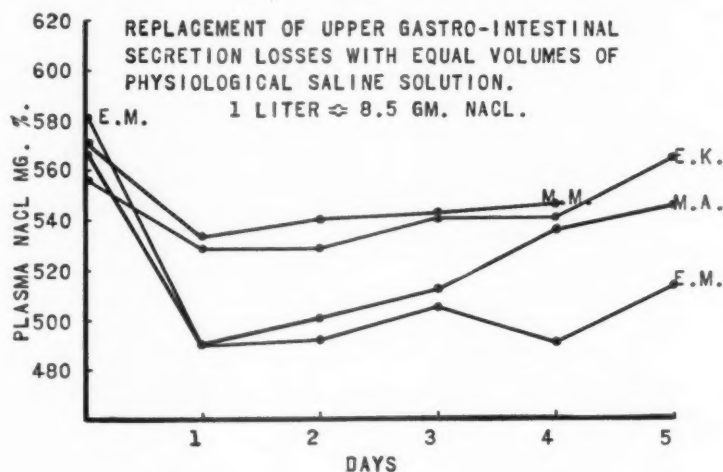
* 465 cc. physiologic saline solution and 425 cc. of blood.

REPLACEMENT OF SODIUM CHLORIDE

patients did not gain in weight; therefore it was assumed that an excessive amount of salt leading to the development of water retention had not been given.

In Table I the data from the patients having their sodium chloride losses replaced with physiologic saline solution are given. The resulting plasma chloride levels are shown graphically in Chart I. Three of the patients (M. M., E. K., and M. A.) maintained a satisfactory plasma chloride level and excreted more than 1 Gm. of sodium chloride in the urine daily. Although the plasma chlorides of the fourth case, E. M., did not fall to a seriously low

CHART I



level, nevertheless, they were definitely below normal. This may be a failure of the volume-for-volume rule, or it may be an example of a patient whose plasma chloride level cannot be brought up to normal.*

In Table II are given the data from the patients whose gastroduodenal secretion losses were replaced by equal volumes of a Ringer's solution containing the equivalent of 7.55 Gm. of sodium chloride per liter.† It will be noted that all of the cases studied (W. W., J. B., A. G., and M. L.) maintained a plasma chloride level above 500 mg. NaCl per 100 cc., but in each the daily urinary excretion of sodium chloride fell below 1 Gm., indicating what we, at this time, believe to be an inadequate excess of salt.

In all except one of the cases of Charts I and II (W. W. of Chart II) there is a definite fall in the plasma chloride concentration during the first 24 hours of the gastro-intestinal fluid drainage, no salt being given during this time. A fairly constant plasma chloride level is thereafter maintained by the volume-for-volume replacement, with a tendency in most cases for the

* This inability to raise the plasma chloride level to normal has been observed in a number of patients.

† Ringer's solution, as made by different laboratories, varies in its composition.

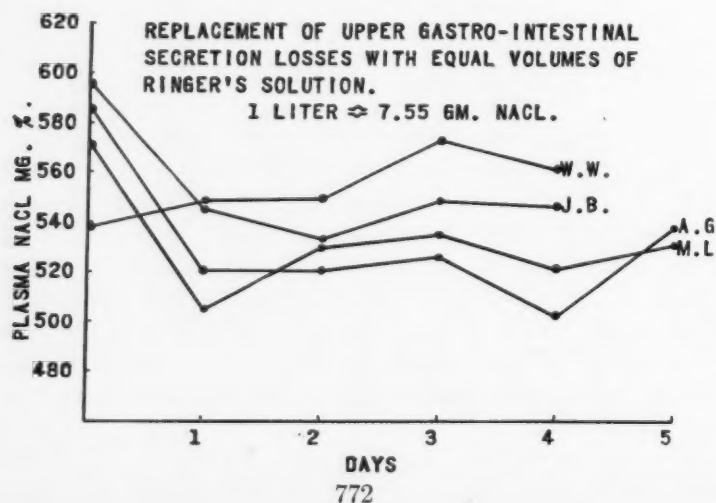
TABLE II

REPLACEMENT OF UPPER GASTRO-INTESTINAL SECRETION LOSSES WITH EQUAL VOLUMES
OF RINGER'S SOLUTION1 Liter \approx 7.55 Gm. NaCl

Patient	24 Hours Ending	Body Weight Kg.	Secretions Lost				Salt Given		Plasma Chlorides Mg. NaCl/ 100 Cc.	NaCl in Urine Gm.
			G.I. Tract Cc.	Bile T-Tube Cc.	Total Volume Cc.	NaCl Gm.	Ringer's Solution Cc.	Salt Content Gm.		
W. W.	1-4	81.77	—	—	—	—	—	—	538	—
	1-5	80.00	960	1,000	1,960	9.97	0	0	548	0.73
	1-6	78.82	290	810	1,100	6.88	2,035	15.36	549	0.54
	1-7	77.81	0	315	315	20.3	1,133	8.55	573	2.10
	1-8	76.96	0	0	0	0	318	2.30	561	0.35
			1,250	2,125	3,375	18.88	3,486	26.21		3.72
J. B.	1-10	73.00	—	—	—	—	—	—	596	—
	1-11	74.50	480	0	480	2.65	0	0	545	3.86
	1-12	73.23	505	0	505	3.30	485	3.66	533	5.19
	1-13	72.33	280	0	280	1.57	522	3.81	548	0.48
	1-14	72.07	0	0	0	0	318	2.40	546	0.32
			1,265	0	1,265	7.52	1,325	9.87		9.85
A. G.	1-10	51.64	—	—	—	—	—	—	586	—
	1-11	—	320	190	510	3.51	800*	4.80	520	1.61
	1-12	49.65	775	180	955	7.18	517	3.90	520	1.02
	1-13	48.78	855	130	985	7.49	945	7.13	526	0.49
	1-14	48.25	860	70	930	5.95	963	7.27	502	0.34
	1-15	49.38	0	150	150	0.88	956	7.22	538	0.27
			2,810	720	3,530	25.01	4,181	30.32		3.73
M. L.	1-24	52.91	—	—	—	—	—	—	571	—
	1-25	52.15	570	290	860	5.60	0	0	505	4.82
	1-26	49.85	540	340	880	4.62	835	6.30	530	0.51
	1-27	49.03	520	250	770	4.41	900	6.79	535	0.17
	1-28	49.76	0	300	300	1.26	787	5.93	521	0.06
	1-29	49.06	0	260	260	1.57	317	2.39	531	0.13
			1,630	1,440	3,070	17.46	2,839	21.41		5.69

* 300 cc. of physiologic saline solution and 500 cc. of blood.

CHART II



REPLACEMENT OF SODIUM CHLORIDE

level to rise on the last day of the study. In an attempt to eliminate this initial drop and thus to maintain the plasma chlorides at a higher level, a series of four patients were given about 1,000 cc. of physiologic saline solution during the first 24 hours of the study. Then, as in the previous cases, the gastro-intestinal secretion losses were replaced volume-for-volume with physiologic saline solution.

TABLE III

REPLACEMENT OF UPPER GASTRO-INTESTINAL SECRETION LOSSES WITH EQUAL VOLUMES OF
PHYSIOLOGIC SALINE SOLUTION PLUS 1,000 CC. PHYSIOLOGIC SALINE SOLUTION DURING
THE FIRST 24 HOURS

1 Liter \approx 8.5 Gm. NaCl

		Secretions Lost					Salt Given			
					Total				Plasma	NaCl
Patient	24 Hours Ending	Body Weight Kg.	G.I. Tract Cc.	Bile T-Tube Cc.	Volume Cc.	NaCl Gm.	Phys. Saline Cc.	Salt Content Gm.	Chlorides Mg. NaCl/ 100 Cc.	in Urine Gm.
T. D.	3-16	—	—	—	—	—	—	—	591	—
	3-17	66.20	120	0	120	0.80	1,050	8.91	561	3.77
	3-18	65.15	110	0	110	0.76	120	1.02	568	1.01
	3-19	63.62	335	0	335	2.27	110	0.93	578	1.33
	3-20	64.03	0	0	0	0	342	2.91	584	0.50
			565	0	565	3.83	1,622	13.77		6.61
E. R.	3-19	46.56	—	—	—	—	—	—	559	—
	3-20	47.29	630	0	630	4.50	1,010	8.60	563	0.36
	3-21	45.30	1,020	0	1,020	7.53	640	5.36	563	0.46
	3-22	44.11	1,890	0	1,890	12.36	1,020	8.65	553	0.13
	3-23	43.21	2,500	0	2,500	19.28	1,920	16.32	530	0.09
	3-24	43.17	3,000	0	3,000	20.76	2,510	21.33	528	0.14
	3-25	42.54	2,330	0	2,330	15.38	3,080	26.20	611	0.41
			11,370	0	11,370	79.81	10,170	86.46		1.59
L. W.	3-22	58.09	—	—	—	—	—	—	579	—
	3-23	59.22	320	0	320	1.96	1,060	9.01	543	3.08
	3-24	57.09	310	0	310	1.82	325	2.70	546	9.02
	3-25	54.45	600	0	600	4.01	314	2.67	504	1.97
	3-26	55.46	0	0	0	0	582	4.91	592	0.64
			1,230	0	1,230	7.79	2,281	19.35		14.71
M. L.	4-12	40.08	—	—	—	—	—	—	602	—
	4-13	40.94	200	220	480	2.87	1,600*	11.41	578	1.33
	4-14	39.08	250	390	640	3.69	486	4.13	568	4.81
	4-15	38.87	0	370	370	2.07	632	5.37	569	1.59
	4-16	37.64	0	390	390	2.17	722	6.14	558	2.12
			510	1,370	1,880	10.80	3,440	17.05		9.85

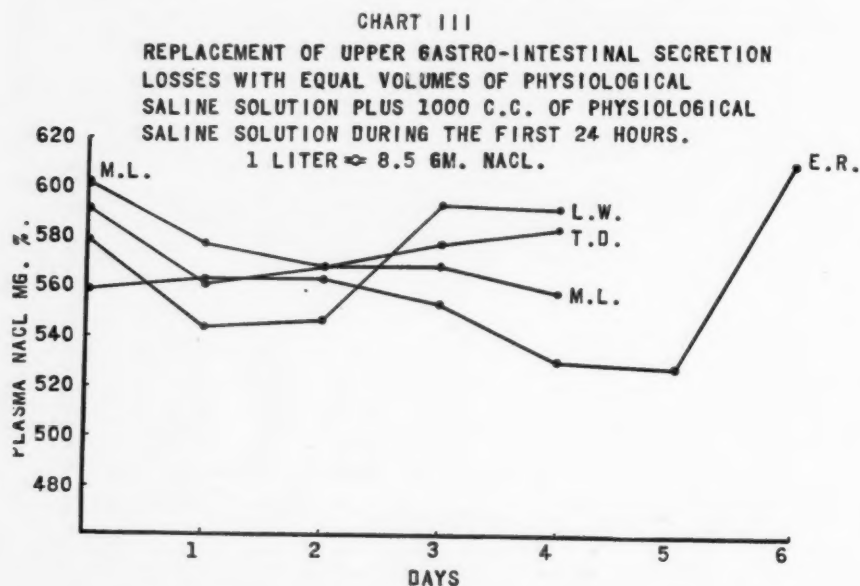
* 1,050 cc. of physiologic saline solution and 550 cc. of blood.

The data from this study are shown in Table III, and graphically presented in Chart III. It will be seen that three of the four cases showed an initial drop in the plasma chloride level in spite of the administration of more than sufficient saline solution to balance the abnormal losses during this period. However, the fall was not as marked as in the cases presented in Tables I and II in which no saline solution was given during the first 24-hour period. Also, the plasma chlorides were maintained at a higher level for the remaining days of the study than was the case for the previous patients.

The maintenance of a satisfactory sodium chloride level in patients losing

significant amounts of gastro-intestinal tract secretions seems to be possible by following the volume-for-volume replacement rule plus the administration of about one liter of physiologic saline solution during the first 24 hours. This latter procedure was added to decrease the initial fall in plasma chlorides which so frequently occurs.

The Restoration of Sodium Chloride.—The idea of putting the sodium chloride needs of patients on a quantitative basis is not new. In 1923, Haden and Orr⁷ suggested that in well advanced intestinal obstruction the patient should be given an initial dose of 1 Gm. of sodium chloride per kg. of body weight. Recently, Falconer and Lyall²⁰ made a further advance in accurate



sodium chloride therapy. They gave known amounts of salt to patients with hypochloremia and determined the resulting rise in the plasma chloride level. From their studies they concluded that "in hypochloremia about 20 grammes (from 15 to 30 grammes) of salt are required on the average to raise the plasma chloride by 100 mg. per 100 cc."

Early in our²¹ investigation of the salt requirements of surgical patients, two simple but fundamental principles became apparent: First, it seemed obvious that the salt needs of a 20 Kg. child must be quite different from those of an adult weighing three times that much. Second, it was thought that if one could determine what percentage of the body salt had been lost, it should be possible to calculate accurately the amount of salt that must be given to restore the body chlorides to normal provided the normal salt content of the body is known.

Various estimations of the total chlorine content of the body have been made. Sherman²² stated the amount to be 0.15 per cent of the body weight.

REPLACEMENT OF SODIUM CHLORIDE

Expressed as sodium chloride, this amounts to 0.248 per cent of the body weight. On this basis, there are 148.8 Gm. of salt in a 60 Kg. individual and 49.6 Gm. in a 20 Kg. child. The importance of body weight in calculations of salt requirements is thus apparent.

As an index of the state of the total body sodium chloride concentration, the value of the plasma chloride level was considered. It has been shown by White and Bridge²³ that a fall in tissue chlorides is directly proportional to the fall in plasma chloride concentration. From this we assume that the plasma chloride concentration can be used as an index of the chloride concentration throughout the body. On this basis, if the plasma chlorides are 20 per cent below normal it is reasonable to consider that about 20 per cent of the body chlorides have been lost. In the previous paragraph are given data that can be used for calculating the total sodium chloride content of the body. With these data and the plasma chloride level one should be able to calculate the grams of salt necessary to be given to a patient with hypochloremia to restore the chlorides to normal, as follows:

(1) Per cent of body salt lost

$$= \frac{\text{normal plasma chlorides} - \text{actual plasma chlorides}}{\text{normal plasma chlorides}} \times 100$$

(2) Total NaCl content of body = 0.248 per cent of body weight (Gm.)

From (1) and (2):

(3) Number of grams of NaCl needed to restore body chlorides to normal

$$= \text{per cent of body salt lost} \times \text{total NaCl content of body}$$

$$= \frac{\text{normal plasma chlorides} - \text{actual plasma chlorides}}{\text{normal plasma chlorides}} \times 100 \times 0.248 \text{ per cent of body weight (Gm.)}$$

$$= \frac{560 - \text{actual plasma chlorides}}{560} \times 0.00248 \times \text{body wt. (Gm.)}$$

Applying the last equation to the example of a 60 Kg. patient admitted to the hospital with a plasma chloride level of 410 mg. NaCl per 100 cc., the formula shows the amount of sodium chloride needed to restore the plasma chloride level to 560 mg. per cent,* *i.e.*, $\frac{560 - 410}{560} \times 0.00248 \times 60,000 = 39.9 \text{ Gm.}$

In order to determine the practical value of this formula, it was applied to a series of individuals with hypochloremia. This group included instances of pyloric and intestinal obstruction, rectal polyp with profuse rectal discharge, paralytic ileus and patients who had been on gastroduodenal suction without accurate replacement of the drainage loss. The patients were given an amount of salt calculated as necessary to restore the plasma chlorides to 560 mg. NaCl per 100 cc. The salt was given intravenously in the form of

* The normal plasma chloride level varies from 560 to 630 mg. per 100 cc. The lower limit was selected for the calculations because many sick patients will not attain a higher level.

physiologic saline or Ringer's solution at the rate of about 500 cc. per hour. During the period of study, 24-hour specimens of urine were collected and the chlorine content determined. If the patient was losing chlorides through some abnormal source during the period of correction of the hypochloremia, these losses were also collected and the salt content determined. These losses in most instances were replaced by the volume-for-volume rule.

TABLE IV

RESTORATION OF BODY CHLORIDES

The formula and clinical calculations are on the basis of a normal plasma chloride concentration of 560 mg. NaCl/100 cc.

Patient	Body Weight Kg.	Initial Plasma Chlorides	Initial Plasma CO ₂ Comb. Power	NaCl Given Gm.	NaCl Lost During Restoration				NaCl Retd. Gm.	Formula Calculation Gm.	Final Plasma Chlorides	Final Plasma CO ₂ Comb. Power	Clin. Calculation Gm.
		Mg. NaCl/100 Cc.	Vol. %		Urine Gm.	Upper G.I. Tract Gm.	Stool Gm.	Total Gm.			Mg. NaCl/100 Cc.	Vol. %	
O. M...	63.3	404	57.6	55.8	7.7	7.3	0	15.0	40.8	43.7	559	52.0	49.6
B. S...	39.0	449	53.6	28.4	9.2	1.2	0	10.4	18.0	19.2	493	67.1	21.6
J. C...	65.5	479	45.7	27.5	2.6	1.4	0	4.0	23.5	23.4	586	44.7	26.5
D. C...	60.4	513	59.8	15.8	1.3	0	0	1.3	14.5	12.6	564	53.9	14.2
D. E...	58.1	345	100.0	100.6	6.2	38.1	?	44.3	56.3	55.3	606	73.0	62.5
L. A. D.	62.0	372	75.0	70.1	14.9	5.5	0	20.4	49.7	51.6	566	60.0	58.3
C. K...	48.9	356	48.0	56.8	0.7	1.1	17.9	19.7	37.1	44.1	528	49.8	49.8
S. L...	21.8	479	—	8.7	0.9	0	0.8	1.7	7.0	7.8	536	—	8.8
L. B. D.	54.0	464	—	25.7	5.4	0.3	0	5.7	20.0	22.9	554	—	25.9
B. M...	34.0	437	—	21.3	0.2	0	0	0.2	21.1	18.5	513	—	20.9
T. J...	72.7	427	59.9	40.6	0.0	0	0	0.0	40.6	42.7	564	48.0	48.3
W. P...	67.7	436	57.3	33.7	1.5	0	0	1.5	32.2	37.1	543	58.3	42.0
J. W...	59.8	447	61.4	26.2	1.1	0	0	1.1	25.1	29.7	546	58.9	33.8
S. T...	73.2	436	67.3	42.8	0.5	0	0	0.5	42.3	39.8	554	56.3	45.4
H. A...	76.9	454	49.5	38.2	0.7	0	0	0.7	37.5	35.6	545	60.7	40.8

The data from this study are shown in Table IV. The close correlation between the amount of salt retained and the amount needed as determined by the formula calculation indicates that the principles of salt replacement previously discussed are sound. In most instances the plasma chloride level determined from 12 to 36 hours after the completion of the saline administration was fairly close to 560 mg. NaCl per 100 cc.

Because of important illustrative points, several cases deserve special comment.

Patient B. S. attained a final plasma chloride level of only 493 mg. per 100 cc. However, this seemed to be the highest level the patient could reach at the time, since she excreted 9.2 Gm. of sodium chloride in the urine and further administration of salt failed to raise the plasma chloride level significantly.

Patient C. K. was moribund when first seen. She had a large rectal polyp associated with frequent watery stools and a profuse rectal discharge, the salt content of which was found to be 5.5 Gm. per liter. Before half of the necessary salt was administered she was awake, and within 24 hours was sitting up in bed and mentally alert. One of the characteristic findings in patients recovering from hypochloremia is a definite euphoria as the plasma chlorides

approach normal. The final plasma chloride level in this patient was only 528 mg. per 100 cc., but it will be noted that the actual salt retention fell 7 Gm. short of the calculated need. This was due to the fact that the patient was losing more salt in the rectal discharge than had been anticipated. The rectal polyp was removed and the patient left the hospital cured. If her moribund state had not been recognized as due to hypochloremia, an operation would never have been possible.

Patient L. A. D. illustrates another instructive point. She was a female, age 82, with a strangulated femoral hernia. Her calculated salt requirement was 51.7 Gm. but by mistake she was given 70.1 Gm. Of the excess 19.4 Gm., 5.5 Gm. were lost in gastroduodenal drainage and 14.9 Gm. were excreted in the urine. In general, small excesses of salt are readily eliminated by the kidneys, but, as others have pointed out, large excesses in sick patients tend to cause edema.

Although the formula was found to be accurate, it is cumbersome, and a search was made for a simpler calculation. Using the formula as a basis, several simple approximations were found, the following being considered the most satisfactory: For each 100 mg. per cent that the plasma chlorides need to be raised to reach the normal of 560 mg. NaCl per 100 cc., the patient should be given 0.5 Gm. of sodium chloride per Kg. of body weight.* Example: For a 60 Kg. patient with a plasma chloride concentration of 410 mg. per cent, the amount of sodium chloride needed is $1.5 \times 0.5 \times 60 = 45.0$ Gm. The figures in the last column in Table IV were calculated on this basis and when compared to the formula calculation they show the adequacy of this simple clinical rule. By its use a slight excess of salt will be given, a desirable feature, without danger.

Discussion.—The clinical syndrome presented by patients with depleted body chlorides is worthy of special comment: They are definitely depressed. There is marked lassitude, weakness, and fatigue. The patient's mentality is dulled, and, in the most severe cases, there may even be stupor and coma. The gastro-intestinal symptoms include, first, a dulling of the sense of taste,²⁴ followed by anorexia, nausea, and vomiting. Muscular cramps also often

* For those who are accustomed to expressing body weight in terms of pounds rather than kilograms, the following rule, which provides for slightly less salt than the clinical calculation, has been formulated: For each 100 mg. per cent that the plasma chlorides need to be raised, the patient should be given 0.2 Gm. of sodium chloride per pound of body weight.

In some laboratories whole blood chlorides rather than plasma chlorides are determined. Using 450 mg. NaCl per 100 cc. as the normal for whole blood, the formula calculation would be:

Gm. NaCl needed

$$= \frac{450 - \text{actual blood chlorides}}{450} \times 0.248 \text{ per cent of body weight (Gm.)}$$

A clinical rule derived from this formula is as follows: For each 100 mg. per cent that the whole blood chlorides need to be raised, the patient should be given 0.6 Gm. of sodium chloride per Kg., or 0.25 Gm. per pound of body weight.

occur. Dehydration, characterized by a dry tongue, sunken eyes, and dry inelastic skin almost invariably accompanies hypochloremia. A low pulse pressure has also been observed; two of our patients presented the clinical picture of shock. Alkalosis with slow respirations and tetany, or inorganic acidosis with deep respirations, may or may not be associated with the chloride depletion.

In the consideration of sodium chloride maintenance and restoration we have dealt with the chloride ion only because, for clinical purposes, its determination is easier than that of sodium. Indirect information concerning the plasma sodium concentration derived from the estimation of the carbon dioxide combining power is always relative to the chloride concentration at that time. For example, gastroduodenal drainage usually contains about equivalent amounts of sodium and chlorine. The loss of a significant volume of these secretions will deplete the body of about equal amounts of sodium and of chlorine, and while the plasma chloride concentration will be definitely lowered, the carbon dioxide combining power will usually be within normal limits. By our use of the plasma chloride determination no implication was intended that chloride is more important than sodium. Undoubtedly the sodium ion is as important, if not more important, than the chloride ion. This was stressed by Gamble and Ross.⁹ In actual surgical practice, the correction of the chloride depletion with sodium chloride also corrects the sodium deficiency which always exists in some degree when gastro-intestinal secretions have been lost. This was emphasized by Gamble,²⁵ who pointed out that sodium chloride given with an abundance of water will correct either alkalosis or inorganic acidosis, the kidneys excreting the unnecessary ion.

It is important to remember that salt is always lost from the body together with water in concentrations which are always less than that of physiologic saline solution. It is apparent, therefore, that salt used for the restoration of body chlorides should be given in isotonic or hypotonic solutions. Hypertonic solutions have the disadvantages that they further dehydrate the patient, and they tend to produce inaccuracies in salt administration by causing salt losses in diarrheal stools. In this study it has been found that physiologic saline or Ringer's solution, given at the rate of 400 to 500 cc. per hour, is retained and corrects sodium chloride deficiency and dehydration. The additional water needed for the daily output of urine and vaporization should be given in the form of 5 per cent glucose in distilled water.

SUMMARY AND CONCLUSIONS

The replacement of sodium chloride lost from the body by vomiting, gastroduodenal drainage, drainage from biliary and intestinal fistulae, diarrhea, wound drainage, and occasionally profuse sweating is a practical problem frequently encountered by the surgeon. Serious depletion of the body sodium chloride will lead to death unless the condition is corrected.

To maintain the normal sodium chloride content of the body in surgical patients losing sodium chloride abnormally while under observation, the fol-

lowing procedure was found to be satisfactory: Administer a volume of physiologic saline solution equal to the volume of the abnormal fluid losses. This procedure has a very practical application when inlying, gastroduodenal suction is employed. In addition to the volume-for-volume rule, in this instance it has been found advisable to give 1,000 cc. of physiologic saline solution during the first day of the drainage period in order to lessen the initial fall in plasma chlorides which commonly occurs.

To restore to normal the sodium chloride content of a patient depleted of these substances, the following clinical rule was found to be effective: For each 100 mg. per cent that the plasma chlorides need to be raised to reach the normal (560 mg. NaCl per 100 cc.) the patient should be given 0.5 Gm. of sodium chloride per Kg. of body weight.

BIBLIOGRAPHY

- ¹ O'Shaughnessy, W. B.: Letter to London Med. Gazette, **9**, 486, 1831-1832.
- ² Hartwell, John A., and Hoguet, J. P.: Experimental Intestinal Obstruction in Dogs with Especial Reference to the Cause of Death and the Treatment by Large Amounts of Normal Saline Solution. *J.A.M.A.*, **59**, 82, 1912.
- ³ MacCallum, W. G., Lintz, Joseph, Vermilye, H. N., Leggett, T. H., and Boas, E.: The Effect of Pyloric Obstruction in Relation to Gastric Tetany. *Bull. Johns Hopkins Hosp.*, **31**, 1, 1920.
- ⁴ Haden, Russell L., and Orr, Thomas G.: The Effect of Sodium Chloride on the Chemical Changes in the Blood of the Dog After Pyloric and Intestinal Obstruction. *J. Exper. Med.*, **38**, 55, 1923.
- ⁵ Haden, Russell L., and Guffey, D. C.: A Case of Pernicious Vomiting of Pregnancy with Low Blood Chlorides and Marked Response to Sodium Chloride Therapy. *Am. J. Obstet. and Gynec.*, **8**, 486, 1924.
- ⁶ Haden, Russell L., and Orr, Thomas G.: The Use of Sodium Chlorid in Treatment of Intestinal Obstruction. *J.A.M.A.*, **82**, 1515, 1924.
- ⁷ Haden, Russell L., and Orr, Thomas G.: Chemical Changes in the Blood of Man After Acute Intestinal Obstruction. *Surg., Gynec. and Obstet.*, **37**, 465, 1923.
- ⁸ Orr, Thomas G., and Haden, Russell L.: Chloride Treatment of Intestinal Obstruction. *Southern Med. J.*, **19**, 300, 1926.
- ⁹ Gamble, James L., and Ross, S. Graham: The Factors in the Dehydration Following Pyloric Obstruction. *J. Clin. Invest.*, **1**, 403, 1925.
- ¹⁰ Dixon, C. F.: The Value of Sodium Chlorid in the Treatment of Duodenal Intoxication. *J.A.M.A.*, **82**, 1498, 1924.
- ¹¹ Raine, Forrester, and Perry, Margaret C.: Intestinal Obstruction—Experimental Studies on Toxicity, Intra-Intestinal Pressure and Chloride Therapy. *Arch. Surg.*, **19**, 478, 1929.
- ¹² Walters, Waltman, Kilgore, Alan M., and Bollman, Jesse L.: Changes in the Blood Resulting from Duodenal Fistula. *J.A.M.A.*, **86**, 186, 1926.
- ¹³ Matas, R.: The Continued Intravenous "Drip." *ANNALS OF SURGERY*, **79**, 643, 1924.
- ¹⁴ Jones, Chester M., and Eaton, Frances B.: Post-Operative Nutritional Edema. *Arch. Surg.*, **27**, 159, 1933.
- ¹⁵ Jones, Chester M., Eaton, Frances B., and White, James C.: Experimental Post-Operative Edema. *Arch. Int. Med.*, **53**, 649, 1934.
- ¹⁶ Ravdin, I. S., and Rhoads, J. E.: Certain Problems Illustrating the Importance of a Knowledge of Biochemistry by the Surgeon. *Surg. Clin. North Amer.*, **15**, 85, 1935.
- ¹⁷ Collier, Frederick A., Dick, Vernon S., and Maddock, Walter G.: Maintenance of Normal Water Exchange with Intravenous Fluids. *J.A.M.A.*, **107**, 1522, 1936.

- ¹⁸ Curphey, W. C., and Orr, T. G.: Edema in Surgical Patients. *Surgery*, **1**, 589, 1936.
- ¹⁹ Dick, Vernon S., Maddock, Walter G., and Collier, Frederick A.: Sodium Chloride Content of Gastro-Intestinal Secretions. *Proc. Soc. Exper. Biol. and Med.*, **37**, 318, 1937.
- ²⁰ Falconer, M. F., and Lyall, A.: The Requirements of Sodium Chloride. *Brit. Med. J.*, 1116, December 4, 1937.
- ²¹ Bartlett, Robert M., Bingham, Dermid L. C., Pedersen, Svend, Maddock, Walter G., and Collier, Frederick A.: Quantitative Studies on the Replacement of Body Chlorides. *Proc. Soc. Exper. Biol. and Med.*, **38**, 89, 1938.
- ²² Sherman, Henry C.: *Chemistry of Food and Nutrition*. Macmillan Co., New York, 242, 1937.
- ²³ White, James C., and Bridge, Edward M.: Loss of Chloride and Water from the Tissues and Blood in Acute High Intestinal Obstruction. *Boston Med. and Surg. J.*, **196**, 893, 1927.
- ²⁴ McCance, R. A.: Experimental Sodium Chloride Deficiency in Man. *Proc. Royal Soc. of London, Series B*, **119**, 245, 1935.
- ²⁵ Gamble, James L.: Dehydration. *New Eng. J. Med.*, **201**, 909, 1929.

DISCUSSION.—DR. THOMAS G. ORR (Kansas City, Kans.): In 1925, Gamble and Ross made the statement that "sodium chloride is the only one of a long list of salts containing both of the ions specifically required for plasma repair." Potassium chloride, calcium chloride, magnesium chloride and ammonium chloride have no value in plasma repair.

The treatment of patients with sodium chloride is a logical and simple type of chemotherapy, being merely a substitution of a body chemical lost by disease. To supply sodium chloride in hypochloremia is comparable to the transfusion of blood for anemia and is just as essential to life.

In addition to the physiologic properties of sodium chloride already mentioned, it may play a rôle in growth, bactericidal power of the blood and maintenance of bowel tone. The prompt response of peristalsis when sodium chloride is given to some patients with distention and hypochloremia leads to the belief that the chloride balance affects the intestinal tone.

We have found, in some recent experiments upon dogs, that sodium chloride is absorbed from the stomach and upper intestine when the jejunum is obstructed 25 cm. below the ligament of Treitz. Animals permitted to drink 0.6 per cent sodium chloride live twice as long as animals drinking tap water. The blood chlorides show relatively little change in the group receiving the salt as compared to those drinking water.

How much water and how much salt to give a sick patient has long been a practical problem. Before the work of Collier and his associates the needs of the patient were estimated by his clinical appearance and the estimation of the blood chlorides. If this is done with understanding, it is quite satisfactory. However, one only needs to observe his own patients and particularly those of his confreres who are not familiar with chloride metabolism to realize the gross inaccuracies of such treatment and the desirability of having some quantitative estimate upon which treatment with water and salt may be based. Everyone who has been interested in sodium chloride therapy in its clinical and experimental aspects realizes the wide margin of safety of this treatment. It is equally well known that too much or too little sodium chloride will cause definite symptoms which mean serious consequences unless corrected. The normal patient or experimental animal will tolerate enormous quantities of sodium chloride without apparent harm, but the sick patient, particularly those who have undergone a starvation period with a reduction

in blood protein, frequently develops an edema which may involve the parenchymatous organs.

From a practical standpoint the method of estimating the quantity of salt needed by equation seems much preferable to measuring the fluids lost and replacing them with the same quantity of physiologic sodium chloride solution. Part of the value of the gastric suction treatment is the pleasure and comfort afforded the patient by drinking water. Any liquid swallowed would upset the balance of measured gastric intake and output. The practical value to the average surgeon of Coller's quantitative method of administering sodium chloride is quite obvious.

DR. FREDERICK A. COLLER (closing): We all realize the importance of restoring the biochemical balance of the sick patient. The present communication is another effort on our part to furnish the clinician with practical working quantitative methods for accomplishing this. Previously, we have reported other studies showing that water losses are the measurable losses from the body, plus an average loss of two liters from the skin and lungs; that enables one to maintain water balance with sufficient accuracy. We have reported our observations showing that the clinical picture of dehydration was produced by the loss of 6 per cent of the body weight in water, and have emphasized the need of replacing fluid and electrolyte losses by the proper fluid; emphasizing the danger of employing saline solutions routinely as a vehicle.

In the present communication we hope to have demonstrated a sound method for replacing sodium chloride losses in an accurate way, simple enough to have an easy clinical application. Hypochloremia is not uncommonly seen in the sick patients in the surgical wards. The symptoms and signs of hypochloremia are not infrequently attributed to the disease causing the loss of body chlorides and often are not recognized as being due primarily to the altered body chemistry. The findings most commonly encountered in this condition are marked lassitude, weakness and a sense of great fatigue. There are dulling of the mentality, drowsiness verging toward stupor and coma, dulling of the sense of taste, anorexia, nausea and vomiting—with occasionally muscle cramps. There are signs of dehydration—dry tongue, sunken eyes, dry inelastic skin and a low pulse pressure. The final proof of the diagnosis rests with the determination of the plasma chloride concentration. Not infrequently alkalosis or inorganic acidosis is also present; alkalosis if the chloride losses are from the stomach. The carbon dioxide combining power as determined shows the acid-base balance and may show alkalosis or inorganic acidosis or be normal with a marked hypochloremia.

In actual surgical practice, the correction of the chloride depletion with sodium chloride almost invariably corrects any disturbance in the acid-base balance. This has been emphasized by Gamble, who pointed out that sodium chloride given with an abundance of water will correct either alkalosis or an inorganic acidosis, the kidneys excreting the unnecessary ion. It is important to remember that salt is always lost from the body together with water in concentrations always less than that of physiologic saline. It is apparent, therefore, that salt used for the restoration of body chlorides should be given in isotonic or hypotonic solutions. In this study, it has been demonstrated that physiologic saline or Ringer's solution given intravenously at the rate of 400 to 500 cc. per hour is retained, and corrects both sodium chloride deficiency and dehydration. The additional water needed for the daily out-

put of urine and for vaporization should be given in the form of 5 per cent glucose in distilled water.

One should emphasize that death may occur when the plasma chlorides fall to approximately half the normal level. Symptoms are usually present when the plasma chlorides fall to a point below 500 mg./100 cc., and that they become serious when they get as low as 400 mg./100 cc. We have been on the lookout for this condition; nevertheless we have nearly lost four patients this year from hypochloremia. In every patient who has lost, or is losing fluid from any part of the gastro-intestinal tract, one must keep careful check of the plasma chlorides, and if they are low they can be replaced with sufficient accuracy by using the formula presented.

BRIEF COMMUNICATION AND CASE REPORT

ACUTE CHOLECYSTITIS IN A *BACILLUS TYPHOSUS* CARRIER*

CHOLECYSTOSTOMY—CHOLECYSTECTOMY—CHOLEDOCHOTOMY

CONSTANTINE J. MACGUIRE, JR., M.D.

NEW YORK

Case Report.—Hosp. No. 78536: A. B., female, age 40, was admitted to the First Medical Division, Bellevue Hospital, February 22, 1937, with a positive blood culture of *Bacillus typhosus* and a typical clinical picture. March 1, 1937, stools were still positive for typhoid bacilli. On March 31, stools were negative for typhoid bacilli. The Board of Health examined them on the 1st, 2nd, 9th and 17th of April, 1937, all of which were negative for typhoid bacilli. The patient was discharged April 20, 1937, apparently cured of an ordinary attack of typhoid fever. She complained only of a slight pain in the right upper quadrant.

She was readmitted April 29, 1937, with a temperature of 104° F.; pulse 120; blood count 10,300, polys. 81 per cent; three plus bile in the urine and marked jaundice of six days' duration; clay colored stools and an icteric index of 72. Previous to readmission she had developed severe pain in the right upper quadrant; fever and chills but no pruritus. Tongue was dry, liver palpable two fingersbreadth below the costal arch, the spleen was palpable and hard; van den Bergh direct, immediate—indirect positive. During the next three days she was very toxic with a temperature as high as 105½° F. On May 4, temperature and pulse commenced to subside. Bile reappeared in the stools and the icterus diminished. Blood culture negative for typhoid and paratyphoid. On May 6, stools were found positive for typhoid bacilli. Duodenal drainage was instituted and about five cubic centimeters of brown bile aspirated 15 minutes after the administration of magnesium sulphate. This showed *Bacillus typhosus* on culture. The icteric index was now 16. Temperature, sepsis and jaundice decreased progressively until May 12, 14 days after admission, when her pulse and temperature were practically normal. The abdomen had become soft except for some spasm in the right upper quadrant, and local tenderness. On May 15, the acute process apparently lighted up again—temperature 104° F.; pulse 120, both of which subsided somewhat during the next few days but the white blood count, of over 20,000, persisted with polys. 93 per cent. There was persistent abdominal rigidity, more marked in the R.U.Q., but the jaundice had not recurred.

Operation.—May 19, 1937: The gallbladder showed evidence of an old chronic cholecystitis. It was shrunken and fibrotic and was the seat of an acute suppurative process. It was buried in a mass of adhesions which involved the stomach and transverse colon and contained many large, hard stones and a moderate amount of very yellow, slimy pus. No attempt was made to detach the adhesions but the main part of the gallbladder was removed leaving the cystic duct and a small pouch of the viscus itself. The stones were removed and the pouch packed with gauze, the peritoneal cavity being walled-off by a rubber dam. The pus from the gallbladder showed a pure culture of *Bacillus typhosus*. She had a smooth convalescence, but there remained a persistent biliary fistula which discharged material which continued to show *Bacillus typhosus* on

* Presented before the New York Surgical Society, October 27, 1937. Submitted for publication January 4, 1938.

culture. The stools also remained positive for *Bacillus typhosus*. She had repeated blood transfusions both before and after the operation.

Because of the persistence of the *Bacillus typhosus* in the stools, she was again operated upon July 7, 1937. The remnants of the gallbladder were buried in a mass of dense adhesions through which ran a fistulous tract which discharged bile. After this had been removed, it was found there were many stones in both the common and right hepatic ducts. The common duct was opened and the stones removed. The right hepatic duct was then evacuated of its calculi and flushed out with saline. A catheter was introduced into the common duct and advanced up into the right hepatic duct. Convalescence was unusually smooth. The first examination of the stool was made six days postoperative and showed no typhoid bacilli, but the discharge from the abdominal wound continued to show a culture of *Bacillus typhosus* until July 27. On August 6, culture from the wound showed only *Staphylococcus aureus*. On August 11, the wound showed only superficial granulations, since which time the stools have remained negative on repeated examinations for typhoid bacilli.

SUMMARY

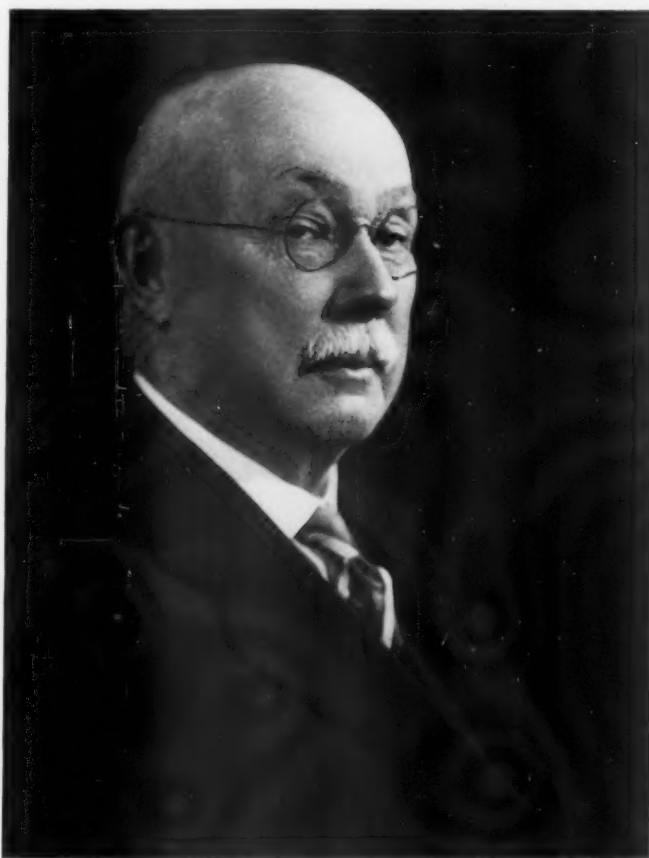
This case is shown because of the following rather unusual features: First, repeatedly negative stools at the end of an attack of typhoid fever. Second, repeatedly positive stools after the development of an acute cholecystitis during convalescence. Third, pure culture of the *Bacillus typhosus* in a suppurative cholecystitis. Fourth, persistence of positive stools after cholecystectomy, probably due to the common and right hepatic ducts containing stones which acted as a focus. Fifth, repeated negative stools following removal of the common and right hepatic duct stones. That these were acting as a focus is indicated by the recovery of *Bacillus typhosus* from the biliary tract for a short time after complete removal of the gallbladder.

MEMOIRS

JOHN JENKINS BUCHANAN

1855-1937

JOHN JENKINS BUCHANAN, whose death occurred August 24, 1937, had one of the most brilliant surgical careers in the history of western Pennsylvania. His success as a surgeon and a teacher of surgery was to a great extent influenced by his background. He was descended from a line of



JOHN JENKINS BUCHANAN, M.D.

Scotch-Irish and Welsh forebears who distinguished themselves as theologians, educators and organizers. His two great-grandfathers served as Revolutionary soldiers, and another of his ancestors was responsible for founding Lafayette College in Pennsylvania, which he served as its President for 14

years, later serving as President of Washington University at Lexington, Virginia, until the Civil War. Doctor Buchanan's grandfather was a well-known clergyman, as was his uncle who distinguished himself as a leading educator of southeastern Ohio.

His father, Dr. James G. Buchanan (1825-1909), established his practice in Wellsville, Ohio, and was appointed Railroad Surgeon when the rails were first laid from Cleveland to Wellsville. For 50 years, until the time of his death, he served as Surgeon to the railroad lines which were incorporated into the great Pennsylvania Railroad System. This service was continuous with the exception of a four year interval when he served his country as Military Surgeon in the Union Army during the Civil War.

Doctor Buchanan was born at Wellsville, Ohio, in 1855, and received his primary education in the public schools there, and later in Allegheny City (now the North Side of Pittsburgh), where the family moved in 1866. He pursued a classical course in the Preparatory Department of the Western University of Pennsylvania (the present University of Pittsburgh), passing on to his collegiate course from which he graduated in 1877, and received his master's degree in 1880. His medical education was received in the University of Pennsylvania at Philadelphia, where he won his M.D. degree in 1881, with the first class required to take a three year course. After serving his internship at the Western Pennsylvania Hospital, he settled down to a general medical and surgical practice and received the appointment as one of the surgeons to the Pennsylvania Railroad Lines west of Pittsburgh. He soon began to operate on every surgical case he felt competent to undertake, many of these operations being performed in private houses, because the patients were generally poor and prejudiced against hospital treatment. He established a surgical clinic at the Pittsburgh Free Dispensary, where his first public surgical work was performed.

In these early years he served for a brief time on the staff of the Allegheny General Hospital, but, in 1891, when he was appointed Surgeon to the Mercy Hospital, his real life work began, for he served that institution faithfully and continuously from that time until his death. In 1921, he was appointed Chief Surgeon and Chief of the General Staff. Aside from his work at Mercy Hospital, he found time to organize the Surgical Departments of the Pittsburgh Hospital and Columbia Hospital, acting as Chief Surgeon of the former institution for nine years, and the latter for six years.

His teaching career began in 1901 with his appointment as Professor of Surgery and Clinical Surgery in the Western Pennsylvania Medical College, now the Medical School of the University of Pittsburgh, and continued with a brief interval until 1936. It is impossible to estimate the influence of his teaching on the development of surgery in western Pennsylvania.

Doctor Buchanan's influence extended to the field of medical literature, to which he was a frequent contributor of papers on surgical subjects. In 1886, he collaborated in the publication of the Pittsburgh Medical Review, with the watchword: "No secret proprietary nor Trade-Mark medicines advertised in

this journal." Rather than break this rule, the editors made up the deficits incurred in its publication from their own scanty earnings. This journal later became the direct progenitor of the Pennsylvania Medical Journal, the present organ of the Medical Society of Pennsylvania. The Library of the Pittsburgh Academy of Medicine was an outgrowth of the Pittsburgh Medical Library which Doctor Buchanan organized in 1891. He was actively interested also in the Library of the Mercy Hospital, and fostered its use among the Staff members.

Doctor Buchanan was an indefatigable worker and a disciplinarian, maintaining his interest and supervision of the affairs of the hospital until he died. His enthusiasm and unswerving loyalty to the ideals of the profession, his mastery of detail, and his keen sense of humor combined to make him an ideal teacher and administrator. He was a true friend whose counsel and advice were sought by his professional brethren, young and old. His greatest relaxation was to pore over old books on special subjects in which he was interested, particularly very old medical works. Although he had no hobby, his greatest pastime was motoring to new places.

Doctor Buchanan was a charter member of the International Society of Surgery, and, in 1911, was elected to Fellowship in the American Surgical Association. He was a founder and life member of the American College of Surgeons, serving on the Board of Governors continuously from 1916 until his death. He was also a member of the Committee of Standards from Pennsylvania from 1916 through 1920, and of the Pennsylvania State Executive Committee which he served as Chairman. In the late 80's and early 90's Doctor Buchanan was Recording Secretary of the Allegheny County Medical Society and, in 1920, was its President.

Besides his wife, Ellen Grier Buchanan, whom he married on June 30, 1887, he leaves two sons to carry on the surgical and professional traditions of the family: Dr. E. P. Buchanan, a surgeon at Mercy Hospital, and John G. Buchanan, a prominent Pittsburgh attorney.

In January, 1936, Doctor Buchanan's colleagues and members of various civic groups joined to pay tribute to him as one of Pittsburgh's most useful and public-spirited citizens. At the testimonial dinner given in his honor, he spoke the words which best expressed his lifelong ambition which he fulfilled so well:

"It will be the pride of my life if, when I pass, I shall leave a coterie of men whose natural abilities I have been able to shape in a surgical way. I know of no higher compliment to any man than the approval and good will of the fellows of his craft."

OTTO C. GAUB.

ALLEN BUCKNER KANAVEL

1874-1938

QUIET, unassuming, friendly, with a genuine and alert interest in his profession and life and a kindly judicial temperament given but to a chosen few, Allen Buckner Kanavel's memory will continue to exert his influence upon



Photo by Du Bois

ALLEN BUCKNER KANAVEL, M.D.

surgical thought and procedures. The contribution he chose to leave to surgery above all else was the surgical training, the stimulus and interest in sur-

gical progress he was able to pass on to his younger associates. This, he felt, would live on after other more scientific contributions had passed.

The son of a Methodist minister, Doctor Kanavel was born September 2, 1874, in Sedgwick, Kansas. After graduating from the college of liberal arts at Northwestern University, he entered the medical school and was graduated with honors, in 1899. He spent six months in Vienna in postgraduate study, and then entered the Cook County Hospital for his internship. Immediately thereafter, he became associated with the department of surgery at Northwestern University Medical School and remained a member of its surgical faculty to the time of his tragic death.

He was impressed, early in his surgical career, with the uncertain and haphazard treatment given to patients with infections of the hand. After ten years of patient, meticulous study of the anatomy of the hand and the prosecution of a wholly original method of investigating the tendon sheaths and fascial spaces of the hand, he published a monograph on the subject, in 1912. This work remains today as his most important contribution to surgical science, and affords a basis for our present and future knowledge of the efficient care of this common and often seriously disabling condition. It is given but to a few to make such a fundamental addition to surgical knowledge.

However, he was introduced into a surgical practice before the day of surgical specialism and was keenly interested in abdominal, neurologic, thyroid and plastic surgery. In at least two of these fields he was a pioneer in Chicago, and through his interest and help stimulated those who came in contact with him to carry on the torch he handed them.

From the inception of Surgery, Gynecology and Obstetrics, he was closely associated in its development and continued to direct its activities. Its contents speak, far more eloquently than words, of the time, effort and thought he gave to it as associate editor and editor. He strove to make it a vital force in aiding the practitioners of surgery in America, those men not associated with teaching centers, to keep abreast of the rapid changes constantly going on in surgical practice.

Apart from the respect and admiration his surgical ability and judgment commanded, he was loved for his constant adherence to the principles of his philosophy of life. He was quick to recognize and listen to the opinions of others regardless of their station or age. He was a great believer in the adage that time heals all difficulties and, therefore, avoided controversy; yet when dilatory tactics, brought about by differences of opinion, threatened a patient's welfare he was quick to act and assume full responsibility for his judgment. He had a simple, homely, charming manner; a shrewd common sense which transcended a more extensive complicated scientific theoretic knowledge, and an unfailing courtesy and kindness. He had a genuine interest in people, particularly the young men in medicine, and he was quick to recognize and to reward merit, without thought of his own personal fortunes or ambitions. With many interests outside his profession he was able to devote himself in his later years to their development. These interests in

geology, astronomy and books were a part of his plan of life formulated many years before his actual retirement from the practice of surgery, although he never lessened his interest in the advancement of surgical thought and teaching. Occupied throughout his life by an intensive study of obvious and practical surgical procedures, he strove to advance the frontiers of medical knowledge; to emphasize that an unselfish service to humanity, personal and professional honesty, the desire to seek new truths, industry, broad culture, judgment and imagination, even more than technical efficiency, are the qualities to be desired by every surgeon, worthy of the name.

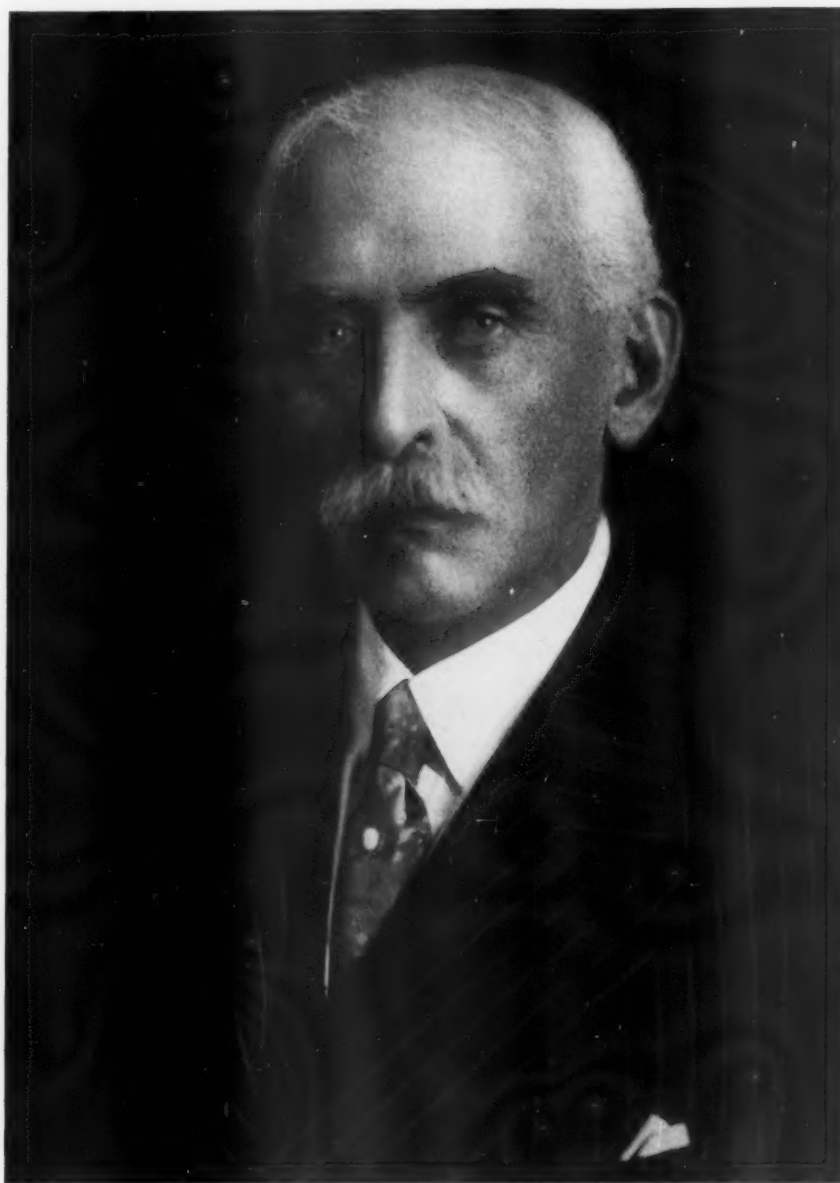
With a mind filled with ideals, a soul possessed of kindness and a sympathetic understanding of all human frailties, he leaves behind him a well-spent life, an example for younger surgeons to emulate and to those of us who loved him, a memory to be revered.

LOYAL DAVIS.

JOSEPH AUGUSTUS BLAKE

1864-1937

FROM the Dean of the College of Physicians and Surgeons, when Doctor Blake was Professor of Surgery, comes this letter.



JOSEPH AUGUSTUS BLAKE, M.D.

"Joseph Augustus Blake was born in 1864 in California where his father was serving as state geologist. Later, when the family moved to New Haven, he naturally, following in his father's footsteps, graduated from Yale College in 1885. He then spent a year in the laboratory of Russell H. Chittenden and, equally naturally, devoted most of his time as a medical student in the anatomical department of the College of Physicians and Surgeons, where he graduated as M.D. in 1888.

"His bent was distinctly scientific by inheritance, environment and education. He served as intern in St. Luke's Hospital and by 1890 was an attending surgeon in St. Luke's and Harlem Hospitals. He continued to develop the scientific features at the college, as a demonstrator under the leadership of the distinguished anatomist George S. Huntington, and always made an academic background to his surgical career. He was assistant to William T. Bull and later full attending surgeon at Roosevelt and Presbyterian Hospitals where he helped to advance the instruction of undergraduates into the surgical wards.

"Doctor Blake always took a broad and catholic view of medical education and hospital development. It was he who first suggested the present site as the best for the newly organized medical center combining the Presbyterian Hospital and Columbia's College of Physicians and Surgeons.

"Doctor Blake as a surgeon showed a complete development of the trained physician. His powers of diagnosis were exact and thorough, both in his methods of eliminating internal medical diseases and in differentiating an existing surgical condition. He always started his operations with a definite diagnosis and discarded the easygoing habit of many colleagues to make his surgical diagnosis with his knife. As a teacher he presented his material in a clear and complete form and left his students to catch up with him. He never approached his subject at their level but usually left it higher than they could reach without personal and individual study on their part."

SAMUEL W. LAMBERT.

Doctor Brewer, closely associated with Doctor Blake in their professional and educational careers, writes as follows:

"As a wise and generous colleague, a helpful and intelligent co-worker, a gifted and resourceful surgeon; and, as an intimate and loyal friend, I have known Dr. Blake, esteemed and admired him for fifty years. . . .

"By a singular coincidence, our professional lives exhibited an unusual parallelism. For nine years we served together as Assistant Demonstrators of Anatomy in the College of Physicians and Surgeons. Both were transferred on the same day to the Surgical Department, as Instructors in Clinical Surgery. Both passed through the various intermediary grades, eventually to become Professors of Surgery in the same institution.

"In hospital positions we also followed similar lines. After serving as Assistant Visiting Surgeons, he at St. Luke's, I at the City and at Mt. Sinai Hospitals, we both were appointed on the same day as Junior Surgeons

to the Roosevelt Hospital, he on the second division with Dr. Bull, I on the first with Dr. Weir. On the retirement of Dr. Bull and Dr. Weir, we both were advanced to the rank of Senior Surgeons. Also we both served, but at different periods, as Attending Surgeons at the Presbyterian Hospital.

"During the World War, we both acted as voluntary surgeons in France, he as Attending Surgeon to the American Ambulance at the Lycée Pasteur in Paris, I at Hospital B, at Juilly, S. et M. about 30 kilometers north of Paris.

"After the United States entered the War, we both were commissioned and served in the A.E.F., as operating surgeons in various military hospitals, and at a later period as Consultants. At the end of the War, each of us retired with the rank of Colonel.

"Doctor Blake made many important contributions to surgical literature, covering his work in clinical surgery, research in the laboratory of experimental surgery, and in surgical pathology.

"As a diligent student and observer, a clever and mechanically-minded technician, he originated better methods of approach in deep seated lesions, new and ingenious methods of avoiding, lessening, and treating surgical shock. He invented new instruments and devices to shorten and make safer operative procedures in gravely debilitated and handicapped patients.

"His wise and sane judgment, his unusual diagnostic skill, his transparent honesty and pleasing personality, won for him many friends and brought to him many patients of all classes, as well as many professional colleagues who relied upon him for help in meeting their difficult and often obscure problems. These qualities and his outstanding early successes, placed him in the foremost rank of American surgeons.

"I have always felt, as have many of his other friends and associates, a sincere regret that Dr. Blake did not publish in full an account of his long experience in military surgery.

"Entering the service of the American Ambulance in Paris, a few weeks after the beginning of hostilities, he served continuously and without interruption until the end of the War.

"His experience in the treatment of battle casualties was probably larger and more extensive than that of any other American surgeon. Realizing as he did the disastrous results which followed the plan adopted by the military authorities during the early months of the War, of transporting the great majority of the grave injuries to hospitals in the rear, often requiring several days without surgical treatment, or with the most inadequate procedures at the first-aid or temporary dressings stations, the grave infections such as those by the streptococcus, gas-producing organism, tetanus, *etc.*; as well as the extensive gangrene that followed delay, where important blood vessels were injured, or impeded circulation which resulted from tourniquets or constricting bandages; as well as the added trauma produced by untreated or badly splinted fractures of the extremities; led him to use all his influence to change these unfortunate conditions, to those in which such grave injuries could receive definite

and adequate surgical treatment before transportation to the hospitals at the rear, a plan which was later adopted by all the Allied Armies.

"A record of the many and great advances which he made and advocated, especially in the treatment of such grave conditions as compound and gravely infected fractures of the extremities, the changed and more scientific methods he devised and employed in injuries of the abdominal, pleural, and cranial cavities, as well as the various changes which he made in splinting, extension, and postural apparatus, would all be of the greatest service to military surgeons in any future war.

"Wholly apart from our professional relations, I always found Dr. Blake a charming and delightful companion on vacational excursions. Our common love of the woods, mountain climbing, fishing and hunting, brought us together on many occasions; and I shall always cherish and remember with unalloyed pleasure our outings together in the Adirondacks and in the Canadian woods."

GEORGE E. BREWER.

Dr. Walton Martin assisted Doctor Blake on his staff at Roosevelt Hospital as an Assistant Attending Surgeon. They were an extraordinarily gifted pair to work for. Writing "of him as I knew him in the days at Roosevelt Hospital," he states, 30 odd years later, that he was "the greatest surgeon I have ever come in contact with."

WALTON MARTIN.

Dr. William C. Clarke, after completing a surgical internship in the New York Hospital, where he worked with many of Doctor Blake's surgical seniors and contemporaries, taught histology at the College of Physicians and Surgeons. He soon came to see the importance of the microscopic study of the tissues in surgical diseases and gave his life to it. It is unnecessary to write of his contributions to Surgical Pathology, for he was its pioneer in the "P. & S." under Doctor Blake. This inspiring teacher pays his tribute.

"In an association of over forty years with the medical men of the College of Physicians and Surgeons and several hospitals associated with the College, from the last years of Stimson, Hartley, Bull, Weir and McBurney, to the present, Dr. Joseph A. Blake stood out, preeminent. There were reasons. Truth was the basis for his evaluation of clinical evidence, not human emotions. He rarely was wrong in a diagnosis. His surgical judgment was referred to as superb. Gifted technically, with the ability to execute ably, his surgical results were of the best. At the same time raised in the highest ideals of his profession, he was 'a real doctor,' and his patients had deep respect for, and confidence in, his opinions, ability and judgment.

"Last summer he had built what proved to be his last shop for his wood working tools. 'Down East' in Maine he personally packed those much loved machines and tools and with equal care unpacked them in their new home in Litchfield. There were thirteen machines, and two thousand and five tools by actual count. Some of them he had devised himself and many he had actually made.

"I never remember his resorting to the so-called 'differential diagnosis method' as a procedure, in which the composite of many patients are invoked having no connection with the single patient before him. He was a reader of evidence in the patient before him. An unusual accuracy of vision of what happened in living man caused people to say it was intuitive and, indeed, he seemed unconsciously aware that a patient did, or did not, have appendicitis, or carcinoma, or some other such disease. Once his mind was made up, he wandered little in probabilities—he acted.

"He was a lover of nature, a biologist, a physician, a surgeon, and always a true scientist."

WILLIAM C. CLARKE.

Doctor Russell knew Doctor Blake as an assistant and associate in his surgical practice. He writes as follows:

"My association with Dr. Blake was, fortunately for me, a very happy and instructive one. He was my quiz master in anatomy, during which time I acted as his prosector.

"From 1901 to 1912 I was closely associated with him in Surgery. He combined, to my way of thinking, more things of the master surgeon than anyone it has been my lot to know. A great anatomist, a good pathologist and a real physicist; added to this he possessed a vast knowledge of surgery, and he had that great gift: Judgment, of what to do and when to do it. 'Corn Field sense' in rugged terms—an invaluable asset. He had an inspiration for planning new operations and applying them at the proper time. He was a great teacher, one of the greatest, in his way of imparting knowledge; one had to listen and absorb, for he never rammed his opinion down one's throat.

"I have always thought of him as one of the great surgeons of history. Last spring he made Surgical Rounds with me at the Roosevelt Hospital, on the Service he had headed with distinction for many years, and he was as keen and as interested as of old."

JAMES I. RUSSELL.

Doctor Whipple, now holding the Professorship of Surgery Doctor Blake held when he first knew him, was appointed to the Presbyterian Hospital and the P. & S. teaching staff by Doctor Blake, and knew his quality as an educator, surgeon, and a friend, as few ever did.

"Of the many outstanding qualities that characterized Doctor Blake as a surgeon's surgeon, the one that stood out constantly was his forthright honesty. He was always his own severest critic and would point out his errors in judgment and technique to us, his admiring apprentices, which we never would have noticed had he not called our attention to them. His influence over the younger group of surgeons working with him was permanent because we all knew he said what he meant and meant what he said. There is no doubt in my mind that his example in this respect played the greatest part in setting a standard of fairness and of rugged honesty in his department, in his hospital

and in his surgical service which has stamped every surgeon who had the privilege of training under him and working with him. It was and is a mark of distinction to be known as a Blake man."

ALLEN O. WHIPPLE.

Surgery, from 1904 to 1914, that I knew was intimately associated with Dr. Joseph A. Blake. So subtle are the essential traits that make a preeminent human character that they well nigh baffle description. To be asked to write of him brings difficulty with the honor.

By heritage and environment, he had an inquisitive, scientific interest in the truths of nature. Like an artist, he was intensely sensitive to impressions. As a genius, he used them to human advantage in a career that was once described as a "daily duel with death." He saw with his eyes and felt with his fingers what other men couldn't. He thought creations and saw essentials that other men didn't.

But there was more than that.

There was manual skill and a love for it. Head and hand went hand in hand and the work of his hands seemed eloquent. Those who watched, and the students he taught, thought so.

Simplicity dominated. Essentials were his aim. It was the point that mattered that counted with Blake. Seldom, in the nature of one man, have versatility and simplicity been so combined. Many a life lost in complexity was saved by this simplicity.

An "extraordinarily puzzling ward case" was once surrounded by an extraordinarily puzzled crowd of visiting surgeons from other parts of this country and Europe. An unusually capable intern had seemingly covered every possible detail of the patient's record in elaborate detail. It was painfully evident that the case remained a mystery to the great group of illustrious clinicians. Every sort of suggestion had been made. Finally, it was Blake's turn. He asked two simple questions. The patient said "yes" and then said "no." The mystery was solved and everyone knew the answer.

Surgery seemed so simple when he did it that it was almost dangerous for young students to watch him. It looked so easy. To the initiated, it was inspiring. Those who helped him kept many choice memories. May I tell you one?

Thirty years ago abdomino-perineal resections for rectal cancer were not done as frequently, nor successfully as today. Blake did almost 20 in a row with no deaths. But there were many hard battles. Some showed wisdom and courage ahead of his time—indeed noteworthy, any time. A difficult one was finished, after three hours' work, one Saturday. Pelvic floor closure was difficult and deep. Sunday noon there was vomiting and all was by no means well. It took courage, plus wisdom, to open the wound and lift a prolapsed loop of gut out of the pelvis—30 hours after so serious an initial procedure. But he did it and the man got well.

Few knew more than he of camping trips, fishing tackle, farm problems,

MEMOIRS

shotguns, bird dogs, wild animals and natural phenomena of varied sort. Few shots of his went wild. He could bag most birds with least powder, noise and smoke. A nicety of skilled technic enabled him to mend motor cars, microscopes and men.

There was great kindness of heart toward patients. Deeply attached, grateful and loyal, they boasted of what he had done for them. Doctors besieged him, wealth wanted him, but a meticulous, personal attention to "ward dressings" was conspicuous. The sick poor were his devoted friends.

Whether peritonitis; herniae; anatomy of the brain; bowel surgery; lung, pleura and heart problems; war wounds or fractures will be most associated with Doctor Blake's name makes no difference. He gave the touch of Midas to them all.

HUGH AUCHINCLOSS.

ROBERT BATTEY GREENOUGH

1871-1937

DR. ROBERT B. GREENOUGH, a member of the American Surgical Association since 1911, died suddenly from cardiac disease February 16, 1937, at the age of 66.



ROBERT BATTEY GREENOUGH, M.D.

Doctor Greenough was born in 1871 in Cambridge, Massachusetts. He entered Harvard College, graduating, cum laude, in 1893, and received the degree of M.D., cum laude, from the Medical School in 1896. He completed his medical education as house officer at the Massachusetts General Hospital and later studied in Vienna, where he became particularly interested in tumor pathology; and although primarily a surgeon, this undoubtedly laid the foundation for his interest in cancer.

On his return to Boston he became the assistant to Dr. John Collins Warren, which position he held until his appointment to the Surgical Staff of the Massachusetts General Hospital. He served the Hospital in various positions until 1931, when he retired with the rank of Visiting Surgeon to become a member of the Consulting Staff.

During the World War he went to France with the First Harvard Unit as Surgeon and executive officers at the American Ambulance in 1915. After the entrance of the United States in the war he was appointed Lieutenant Commander and was in charge of the Surgical Service at the Naval Hospital in Chelsea.

He was actively connected with the Harvard Medical School, receiving the appointment of Assistant in Surgery in 1901 and Assistant Professor in 1910, which position he held until 1931.

His executive ability was recognized by all and he was called upon to fill many responsible positions. Among the positions held by him the following may be mentioned:

- President, American College of Surgeons, 1934-1935
- President, Massachusetts Medical Society, 1929-1931
- President, Boston Surgical Society, 1928-1930
- Secretary, American Surgical Association, 1922-1926
- President, American Society for the Control of Cancer at the time of his death.

The study of tumors may be said to have been his life's work, and his first contribution to literature, *Plummer's Bodies in Cancer*, was published in 1901.

When Doctor Greenough became associated with Doctor Warren, the latter was Co-Trustee with Doctor Oliver of the Caroline Brewer Croft Fund for Cancer Research. In 1909 this fund was combined with others and placed in the hands of the Cancer Commission of Harvard University. Doctor Greenough was appointed Secretary of the Commission. In 1912 he took an active part in the building and organization of the staff of the Collis P. Huntington Memorial Hospital for Cancer Research, and acted as Director of the Commission and Surgeon to the Hospital from 1915 to 1929. While recognizing the value of research work he saw no reason why members of the present generation who might be suffering from malignant disease should not have the advantage of the best known diagnostic facilities and treatment. The Consultation Cancer Clinics developed under him at the Huntington Memorial and Massachusetts General Hospitals have served as

models for those advocated by the American College of Surgeons and universally adopted throughout the country.

Besides his professional ability as a surgeon and executive, he had the unusual faculty of making no enemies. If discussion arose in a committee or other meeting he would consider fairly the dissenter's point of view and bring the matter under discussion to an amicable settlement satisfactory to all concerned.

Outside of his profession he was a delightful companion and had a host of friends. It was the writer's privilege to have known him and his family intimately, and a trip or vacation taken with him was a delightful experience.

His death has been keenly felt by his medical colleagues and his many other friends.

CHANNING C. SIMMONS.

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